WEST

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Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Term:

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Generate: ○ **Hit List** ◉ **Hit Count** ○ **Image**

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Search History

Today's Date: 1/15/2002

DB Name	Query	Hit Count	Set Name	
USPT,PGPB	Fusobacterium varium or f varium	36	L1	Bib only
USPT,PGPB	colitis	4594	L2	
USPT,PGPB	s l1 same l2	0	L3	
USPT,PGPB	l1 same l2	0	L4	
USPT,PGPB	l1 and l2	0	L5	
USPT,PGPB	vaccin$	17885	L6	
USPT,PGPB	l1 same l6	0	L7	
USPT,PGPB	l1 and l6	3	L8	
USPT,PGPB	antigen	40419	L9	
USPT,PGPB	l1 same l9	0	L10	

USPT,PGPB	l1 and l9	3	L11	
USPT,PGPB	l11 not l8	0	L12	same hits
USPT,PGPB	antibody	55158	L13	
USPT,PGPB	l1 same l13	0	L14	
USPT,PGPB	l1 and l13	4	L15	
USPT,PGPB	l15 not l8	1	L16	one new hit
USPT,PGPB	adhesin	437	L17	
USPT,PGPB	adhesion	150032	L18	
USPT,PGPB	l17 or l18	150157	L19	
USPT,PGPB	l1 same l19	0	L20	
USPT,PGPB	l1 and l19	2	L21	
USPT,PGPB	l21 not (determining relative abundance)/ti	2	L22	
USPT,PGPB	l21 not (determining relative abundance).ti.	1	L23	
USPT,PGPB	ulcer$	17271	L24	
USPT,PGPB	l1 same l24	2	L25	
USPT,PGPB	l25 not (l8 or l21)	0	L26	same hits
USPT,PGPB	model	303766	L27	
USPT,PGPB	l2 same l27	263	L28	
USPT,PGPB	l28 same l1	0	L29	
USPT,PGPB	l28 and l1	0	L30	no hits
USPT,PGPB	butryic acid or butyrate	28608	L31	
USPT,PGPB	l28 same l31	0	L32	
USPT,PGPB	l28 and l31	8	L33	
USPT,PGPB	animal same l27	18596	L34	
USPT,PGPB	animal with l27	14934	L35	
USPT,PGPB	l35 with l2	36	L36	
USPT,PGPB	l36 and l31	1	L37	
USPT,PGPB	(l1 and l24).ti.	0	L38	
USPT,PGPB	diagnos$	106386	L39	
USPT,PGPB	l2 with l24	3702	L40	
USPT,PGPB	l40 same l39	458	L41	
USPT,PGPB	l41 and l1	0	L42	
USPT,PGPB	l41 and l31	6	L43	
USPT,PGPB	l31 same l41	0	L44	
USPT,PGPB	l33 not l43	8	L45	
USPT,PGPB	l1 same l39	0	L46	

USPT,PGPB	11 and 139	8	L47
USPT,PGPB	147 not (18 or 116 or 133 or 137 or 143 or 145)	6	L48
USPT,PGPB	Fusobacterium (Broaden Search)	641	L49
USPT,PGPB	149 not 11 (Remove hits already looked at.	607	L50
USPT,PGPB	150 same 16	31	L51
USPT,PGPB	150 with 16	10	L52
USPT,PGPB	150 same 12	0	L53
USPT,PGPB	150 and 12	31	L54
USPT,PGPB	150 and 140	13	L55
USPT,PGPB	150 same 135	0	L56
USPT,PGPB	150 and 135	54	L57
USPT,PGPB	157 and 131	5	L58 False hits
USPT,PGPB	155 not (DNA-binding molecules).ti.	6	L59
USPT,PGPB	159 not (DNA binding molecules).ti.	5	L60

WEST

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Search Results - Record(s) 1 through 36 of 36 returned.

☐ 1. Document ID: US 6331550 B1

L1: Entry 1 of 36 File: USPT Dec 18, 2001

US-PAT-NO: 6331550
DOCUMENT-IDENTIFIER: US 6331550 B1
TITLE: Methods of use of quinolone compounds against anaerobic pathogenic bacteria

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 6204051 B1

L1: Entry 2 of 36 File: USPT Mar 20, 2001

US-PAT-NO: 6204051
DOCUMENT-IDENTIFIER: US 6204051 B1
TITLE: Apparatus and method for growing anaerobic microorganisms

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 6107033 A

L1: Entry 3 of 36 File: USPT Aug 22, 2000

US-PAT-NO: 6107033
DOCUMENT-IDENTIFIER: US 6107033 A
TITLE: Methods and materials for determining relative abundance of microorganisms in mixed populations

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 4. Document ID: US 6056978 A

L1: Entry 4 of 36 File: USPT May 2, 2000

US-PAT-NO: 6056978
DOCUMENT-IDENTIFIER: US 6056978 A
TITLE: Use of hyperimmune milk to prevent suppression of T-lymphocyte production

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 5955344 A

L1: Entry 5 of 36 File: USPT Sep 21, 1999

US-PAT-NO: 5955344
DOCUMENT-IDENTIFIER: US 5955344 A
TITLE: Apparatus and method for growing anaerobic microorganisms

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 6. Document ID: US 5916781 A

L1: Entry 6 of 36 File: USPT Jun 29, 1999

US-PAT-NO: 5916781
DOCUMENT-IDENTIFIER: US 5916781 A
TITLE: Method for producing D-tryptophan

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 7. Document ID: US 5830746 A

L1: Entry 7 of 36 File: USPT Nov 3, 1998

US-PAT-NO: 5830746
DOCUMENT-IDENTIFIER: US 5830746 A
TITLE: Apparatus and method for growing anaerobic microorganisms

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 8. Document ID: US 5700787 A

L1: Entry 8 of 36 File: USPT Dec 23, 1997

US-PAT-NO: 5700787
DOCUMENT-IDENTIFIER: US 5700787 A
TITLE: Capsular polysaccharide immunomodulator

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 9. Document ID: US 5578470 A

L1: Entry 9 of 36 File: USPT Nov 26, 1996

US-PAT-NO: 5578470
DOCUMENT-IDENTIFIER: US 5578470 A
TITLE: Method for preparing thiol compounds with bacterial .beta.-lyase

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 10. Document ID: US 5219842 A

L1: Entry 10 of 36 File: USPT Jun 15, 1993

US-PAT-NO: 5219842
DOCUMENT-IDENTIFIER: US 5219842 A
TITLE: Method of improving intestinal floras

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 11. Document ID: US 5182194 A

L1: Entry 11 of 36 File: USPT Jan 26, 1993

US-PAT-NO: 5182194
DOCUMENT-IDENTIFIER: US 5182194 A
TITLE: Method for preparing p-mentha-8-thiol-3-one

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 12. Document ID: US 5116832 A

L1: Entry 12 of 36 File: USPT May 26, 1992

US-PAT-NO: 5116832
DOCUMENT-IDENTIFIER: US 5116832 A
TITLE: Penem derivatives production and use thereof

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 13. Document ID: US 5045458 A

L1: Entry 13 of 36 File: USPT Sep 3, 1991

US-PAT-NO: 5045458
DOCUMENT-IDENTIFIER: US 5045458 A
TITLE: Antibiotics PB-6042S

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 14. Document ID: US 5043450 A

L1: Entry 14 of 36 File: USPT Aug 27, 1991

US-PAT-NO: 5043450
DOCUMENT-IDENTIFIER: US 5043450 A
TITLE: 8-alkoxyquinolonecarboxylic acid and salts thereof

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 15. Document ID: US 5019513 A

L1: Entry 15 of 36 File: USPT May 28, 1991

US-PAT-NO: 5019513
DOCUMENT-IDENTIFIER: US 5019513 A
TITLE: Anti-bacterial T-cell factor

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 16. Document ID: US 4997829 A

L1: Entry 16 of 36 File: USPT Mar 5, 1991

US-PAT-NO: 4997829
DOCUMENT-IDENTIFIER: US 4997829 A
TITLE: Penem compounds, and use thereof

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 17. Document ID: US 4980470 A

L1: Entry 17 of 36 File: USPT Dec 25, 1990

US-PAT-NO: 4980470
DOCUMENT-IDENTIFIER: US 4980470 A
TITLE: 8-alkoxyquinolonecarboxylic acid and salts thereof

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 18. Document ID: US 4904470 A

L1: Entry 18 of 36 File: USPT Feb 27, 1990

US-PAT-NO: 4904470
DOCUMENT-IDENTIFIER: US 4904470 A
TITLE: Antibiotic F-0769, process for its production, and its use as a growth accelerating and feed efficiency increasing agent and as an antitumour agent

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 19. Document ID: US 4894458 A

L1: Entry 19 of 36 File: USPT Jan 16, 1990

US-PAT-NO: 4894458
DOCUMENT-IDENTIFIER: US 4894458 A
TITLE: Quinolonecarboxylic acid derivatives and their preparation

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 20. Document ID: US 4864023 A

L1: Entry 20 of 36 File: USPT Sep 5, 1989

US-PAT-NO: 4864023
DOCUMENT-IDENTIFIER: US 4864023 A
TITLE: Pyrido(3,2,1-IJ)-1,3,4-benzoxadiazine derivatives

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 21. Document ID: US 4826982 A

L1: Entry 21 of 36 File: USPT May 2, 1989

US-PAT-NO: 4826982
DOCUMENT-IDENTIFIER: US 4826982 A
TITLE: Quinolonecarboxylic acid derivatives

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 22. Document ID: US 4801584 A

L1: Entry 22 of 36 File: USPT Jan 31, 1989

US-PAT-NO: 4801584
DOCUMENT-IDENTIFIER: US 4801584 A
TITLE: Pyrido(3,2,1-IJ)-1,3,4 benzoxadiazine derivatives

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 23. Document ID: US 4791118 A

L1: Entry 23 of 36 File: USPT Dec 13, 1988

US-PAT-NO: 4791118
DOCUMENT-IDENTIFIER: US 4791118 A
TITLE: Quinolonecarboxylic acid derivatives and their preparation

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 24. Document ID: US 4782045 A

L1: Entry 24 of 36 File: USPT Nov 1, 1988

US-PAT-NO: 4782045
DOCUMENT-IDENTIFIER: US 4782045 A
TITLE: Promoting the proliferation of intestinal bifidobacteria

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 25. Document ID: US 4759928 A

L1: Entry 25 of 36 File: USPT Jul 26, 1988

US-PAT-NO: 4759928
DOCUMENT-IDENTIFIER: US 4759928 A
TITLE: Antibiotic: Treponemycin

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 26. Document ID: US 4753953 A

L1: Entry 26 of 36 File: USPT Jun 28, 1988

US-PAT-NO: 4753953
DOCUMENT-IDENTIFIER: US 4753953 A
TITLE: Pyridonecarboxylic acid derivatives and antibacterial pharmaceutical compositions thereof

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 27. Document ID: US 4599418 A

L1: Entry 27 of 36 File: USPT Jul 8, 1986

US-PAT-NO: 4599418
DOCUMENT-IDENTIFIER: US 4599418 A
TITLE: Benzoquinolizine carboxylic acid derivatives, and process for preparation thereof

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 28. Document ID: US 4533631 A

L1: Entry 28 of 36 File: USPT Aug 6, 1985

US-PAT-NO: 4533631
DOCUMENT-IDENTIFIER: US 4533631 A
TITLE: Fermentation process for preparation of antibiotic Bu-2517

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 29. Document ID: US 4409210 A

L1: Entry 29 of 36 File: USPT Oct 11, 1983

US-PAT-NO: 4409210
DOCUMENT-IDENTIFIER: US 4409210 A
TITLE: Antibiotic compound

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 30. Document ID: US 4355111 A

L1: Entry 30 of 36 File: USPT Oct 19, 1982

US-PAT-NO: 4355111
DOCUMENT-IDENTIFIER: US 4355111 A
TITLE: Microorganism culturing device

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 31. Document ID: US 4260683 A

L1: Entry 31 of 36 File: USPT Apr 7, 1981

US-PAT-NO: 4260683
DOCUMENT-IDENTIFIER: US 4260683 A
TITLE: Process for producing antibacterial agents

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 32. Document ID: US 4250170 A

L1: Entry 32 of 36 File: USPT Feb 10, 1981

US-PAT-NO: 4250170
DOCUMENT-IDENTIFIER: US 4250170 A
TITLE: Antibacterial agents Bu-2349A and B and method of using same

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 33. Document ID: US 4235964 A

L1: Entry 33 of 36 File: USPT Nov 25, 1980

US-PAT-NO: 4235964
DOCUMENT-IDENTIFIER: US 4235964 A
TITLE: Method for testing and identifying microorganisms

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 34. Document ID: US 4199515 A

L1: Entry 34 of 36 File: USPT Apr 22, 1980

US-PAT-NO: 4199515
DOCUMENT-IDENTIFIER: US 4199515 A
TITLE: Novel lonomycin derivatives

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 35. Document ID: US 4181574 A

L1: Entry 35 of 36 File: USPT Jan 1, 1980

US-PAT-NO: 4181574
DOCUMENT-IDENTIFIER: US 4181574 A
TITLE: Production of antibiotics by fermentation of novel strains of Micropolyspora caesia

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 36. Document ID: US 4169096 A

L1: Entry 36 of 36 File: USPT Sep 25, 1979

US-PAT-NO: 4169096
DOCUMENT-IDENTIFIER: US 4169096 A
TITLE: Antibiotic compounds

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

Generate Collection

Term	Documents
FUSOBACTERIUM.USPT,PGPB.	623
FUSOBACTERIUMS	0
FUSOBACTERIA.USPT,PGPB.	37
FUSOBACTERIAS	0
VARIUM.USPT,PGPB.	86
VARIUMS	0
VARIA.USPT,PGPB.	231
VARIAS	0
F.USPT,PGPB.	1018497
FS.USPT,PGPB.	12608
((FUSOBACTERIUM ADJ VARIUM) OR (F ADJ VARIUM)).USPT,PGPB.	36

Display 36 Documents, starting with Document: 36

Display Format: TI Change Format

WEST

Generate Collection

Search Results - Record(s) 1 through 3 of 3 returned.

☐ 1. Document ID: US 6056978 A

L8: Entry 1 of 3 File: USPT May 2, 2000

US-PAT-NO: 6056978
DOCUMENT-IDENTIFIER: US 6056978 A

TITLE: Use of hyperimmune milk to prevent suppression of T-lymphocyte production

DATE-ISSUED: May 2, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Beck; Lee R.	Lebanon	OH		
Ishida; Atsunori	Honjo			JPX
Yoshikai; Yasunobu	Higashiku			JPX
Murosaki; Shinji	Nara			JPX
Kubo; Chiharu	Hakata-ku			JPX
Hidaka; Yoshio	Tokyo			JPX
Nomoto; Kikuo	Higashi-ku			JPX

US-CL-CURRENT: 424/535; 424/150.1, 424/278.1, 514/878

ABSTRACT:

The invention relates to the use of hyperimmune milk derived from milk producing animals hyperimmunized with bacterial antigens including intestinal bacteria. The present hyperimmune milk effectively prevents the decline of immunological functions observed in aging or immunocompromised animals and prevents the translocation of indigenous enteric bacteria from the GI tract of immunocompromised or aged animals, thereby preventing indigenous infection. More specifically, the present hyperimmune milk is administered to an animal in an amount sufficient to effectively prevent translocation of indigenous enteric bacteria in, delay the onset of, lower the rate of, or restore the declining immune functions of, aging or otherwise immunocompromised animals.

10 Claims, 10 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 9

L8: Entry 1 of 3 File: USPT May 2, 2000

DOCUMENT-IDENTIFIER: US 6056978 A
TITLE: Use of hyperimmune milk to prevent suppression of T-lymphocyte production

DEPR:
The present invention is based in part on the discovery that when a milk-producing animal such as a bovid is brought to a state of hyperimmunization with a vaccine containing intestinal bacteria, the animal will produce milk, which contains supranormal levels of IgG against intestinal bacteria. Oral administration of this hyperimmune milk suppresses the decline of immunological functions associated with advanced physiological age and/or immune senescence

and/or decline observed in an immunocompromised animal. By the term "supranormal" is intended levels in excess of that found in milk from non-hyperimmunized animals.

DEPR:
Step 1: Any intestinal antigens or combination of intestinal antigens may be employed. The critical point in this step is that the intestinal antigen(s) must be capable, not only of inducing immune and hyperimmune states in the milk-producing animal, but also of producing supranormal levels of IgG against intestinal bacteria in the hyperimmune milk. One preferred vaccine is a mixture of polyvalent bacterial antigens, referred to as Series 100 vaccine, described in detail in Example 2.

DEPR:
Step 2: The antigen(s) of Step 1 can be administered to the milk-producing animal in any method that causes sensitization. In one method, a vaccine composed of antigen derived from 1.times.10.sup.6 to 1.times.10.sup.20, preferably 10.sup.8 to 10.sup.10, most preferably 2.times.10.sup.8, heat-killed bacteria is administered by intramuscular injection. However, other methods such as intravenous injection, intraperitoneal injection, rectal suppository, or oral administration may be used.

DEPR:
Step 3: It is necessary to determine whether or not the milk-producing animal has become sensitive to the intestinal-bacteria antigen. There are a number of methods known to those skilled in the art of immunology to test for sensitivity (Methods in Immunology and Immunochemistry, William, C. A., and Chase, W. M., Academic Press, New York, vols. 1-5 (1975)). The preferred method is to use a polyvalent vaccine comprising multiple intestinal-bacteria species as the antigen and to test for the presence of agglutinating antibodies in the serum of the animal before and after challenge with the vaccine. The appearance of milk antibodies after immunization with the vaccine indicates sensitivity; at this point it is possible to proceed to step 4.

DEPR:
Step 4: This involves the induction and maintenance of the hyperimmune state in the sensitized animal. This is accomplished by repeated booster administration at fixed time intervals of the same polyvalent vaccine that was used to achieve the primary sensitization. A two-week booster interval is optimal for polyvalent bacterial antigens. However, it is necessary to ensure that the animal does not pass from a hyperimmune state to a state of immune tolerance to the antigen.

DEPR:
In a preferred embodiment, hyperimmunization of the milk-producing animal may be achieved by a single administration of microencapsulated vaccine, prepared as described in detail in Example 2. The advantage of the controlled release form of hyperimmunization is that the constant exposure to the antigen ensures that the animal remains in the hyperimmune state.

DEPR:
Bacteria or bacteria antigens thereof, suitable for use in the vaccine of the present invention include intestinal bacteria of humans or other animals. Such suitable bacteria include, for example, the following:

DEPR:
Any bacteria present in the intestinal tract of humans or animals, is suitable for use in the vaccine of the present invention. The selection of suitable bacteria from the above listed bacteria is within the knowledge of one of ordinary skill in the art. The selection of other bacteria not expressly set forth above is also within the knowledge of one of ordinary skill in the art.

DEPR:
The media-free bacterial suspension was heat-killed by placing the suspension in a glass flask in an 80.degree. C. water bath overnight. The viability of the broth culture was tested with a small amount of heat-killed bacteria. Broth was

inoculated with heat-killed bacteria, incubated at 37+ C. for five days and checked daily for growth, as the bacteria have to be killed for use in the vaccine.

DEPR:
Cows were given daily injections of 5 ml samples of the polyvalent liquid vaccine. Antibody (IgG) titer levels for the injected cattle were determined periodically by using an enzyme-linked immunoassay for bovine antibody against the polyvalent antigen.

DEPC:
Preparation of S-100 Vaccine

DEPV:
Fusobacterium including F. gonidiaformans (ATCC No.: 25563); FJ. mortiferum (ATCC Nos. 9817; human: 25557); F. naviforme F(ATCC No.: human: 25832); F. necrogenes (ATCC No.: duck/25556); F. necrophorum (ATCC Nos.: 25286; sheep/27852); F. nucleatum (ATCC Nos.: 10953, 23726; human: 25586); F. plauti (ATCC No.: 29863); F. prausnitzii (ATCC No.: human: 27766); F. russi (ATCC No.: cat/25583); F. symbiosum; F. varium (ATCC Nos.: 8501, 27725);

CLPV:
orally administering to said animal hyperimmune milk, in an amount and for a time sufficient to increase the in vitro responsiveness to mitogens of said MLN cells to Phaseolus vulgaris agglutinin, wherein said hyperimmune milk is prepared from a cow immunized with an intestinal-bacteria-containing vaccine, said intestinal-bacteria containing vaccine comprising Staph. aureus, Staph. epidermis, Strep. pyogenes, A Type 1, Strep. pyogenes, A Type 3, Strep. pyogenes, A Type 5, Strep. pyogenes, A Type 8, Strep. pyogenes, A Type 12, Strep. pyogenes, A Type 14, Strep. pyogenes, A Type 18, Strep. pyogenes, A Type 22, Aerobacter aerogenes, E. coli, Salmonella enteritidis, Pseudomonas aeruginosa, Klebsiella pneumoniae, Salmonella typhimurium, Haemophilus influenzae, Strep. mitis. Proteus vulgaris, Shigella dysenteriae, Strep. pneumoniae, Propionibacter acnes (anaerobe) Strep. sanguis, Strep. salvarius, Strep. mutans and Strep. agalactiae bacteria.

CLPV:
orally administering to said animal hyperimmune milk in an amount and for a time sufficient to increase the proliferative response of said MLN cells to allogeneic spleen cells in vitro, wherein said hyperimmune milk is prepared from a cow immunized with an intestinal-bacteria-containing vaccine, said intestinal-bacteria containing vaccine comprising Staph.

CLPV:
orally administering to said animal hyperimmune milk in an amount and for a time sufficient to increase said CD.sup.+ levels, wherein said hyperimmune milk is prepared from a cow immunized with an intestinal-bacteria-containing vaccine, said intestinal-bacteria containing vaccine comprising Staph. aureus, Staph. epidermis, Strep. pyogenes, A Type 1, Strep. pyogenes, A Type 3, Strep. pyogenes, A Type 5, Strep. pyogenes, A Type 8, Strep. pyogenes, A Type 12, Strep. pyogenes, A Type 14, Strep. pyogenes, A Type 18, Strep. pyogenes, A Type 22, Aerobacter aerogenes, E. coli, Salmonella enteritidis, Pseudomonas aeruginosa, Klebsiella pneumoniae, Salmonella typhimurium, Haemophilus influenzae, Strep. mitis. Proteus vulgaris, Shigella dysenteriae, Strep. pneumoniae, Propionibacter acnes (anaerobe) Strep. sanguis, Strep. salvarius, Strep. mutans and Strep. agalactiae bacteria.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

2. Document ID: US 5700787 A

L8: Entry 2 of 3 File: USPT Dec 23, 1997

US-PAT-NO: 5700787
DOCUMENT-IDENTIFIER: US 5700787 A

TITLE: Capsular polysaccharide immunomodulator

DATE-ISSUED: December 23, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Tzianabos; Arthur O.	Reading	MA		
Onderdonk; Andrew B.	Westwood	MA		
Kasper; Dennis L.	Newton Center	MA		

US-CL-CURRENT: 514/54; 424/831, 514/55, 514/56, 514/61

ABSTRACT:

Methods and products for protecting against abscess formation associated with surgery, trauma or diseases that predispose the host to abscess formation are provided. Methods for forming immunomodulators and pharmaceutical compositions relating thereto also are provided. The products useful in the invention are polysaccharides including a repeat unit having a positively charged free amino group and a negatively charged group. The preferred polysaccharide is B. fragilis capsular polysaccharide A.

13 Claims, 0 Drawing figures Exemplary Claim Number: 1

L8: Entry 2 of 3 File: USPT Dec 23, 1997

DOCUMENT-IDENTIFIER: US 5700787 A
TITLE: Capsular polysaccharide immunomodulator

BSPR:
It has been impractical to immunize patients against abscess formation such as in the case of intra-abdominal surgery. This traditional approach to treatment or prevention is not possible because there simply are too many strains of bacteria capable of causing abscess formation, and protection against one would not confer protection against another. It furthermore is unsettled whether vaccination and consequent induction of an immune response would confer adequate protection against abscess formation by any particular bacterium. There also exist problems and dangers associated with administering live or attenuated strains of bacteria to humans, further discouraging efforts to produce vaccines containing a large number of different bacteria.

BSPR:
Although subcutaneous administration of either B. fragilis or CPC is sufficient to protect animals against abscess formation subsequent to challenge with B. fragilis or CPC, neither conferred immunity against other bacterial strains, as was expected. They therefore have no use as a "vaccine" for abscess formation caused by the multitude of organisms normally found in the colon.

BSPR:
It has been discovered that certain polysaccharides can be used to stimulate host T cells and induce protection against numerous bacteria. This protective effect is T cell-dependent and not mediated by a humoral antibody response. As such, administration of the preparations of the invention is not "vaccination" and the preparations are not "vaccines" which mediate protection that is specific to bacteria expressing the immunizing antigen.

DEPR:
B. fragilis NCTC 9343 and ATCC 23745, B. distasonis ATCC 8503, and Fusobacterium

varium ATCC 8501 were originally obtained from the National Collection of Type Cultures (London, England) or the American Type Culture Collection (Bethesda, MD). B. thetaiotaomicron 5482 and Enterococcus faecalis 2603 strains were obtained from stock culture collections of the Channing Laboratory, Brigham and Women's Hospital (Boston, Mass.). Microorganisms were stored at -80.degree. F. in peptone-yeast or brain heart infusion broth until used, and grown anaerobically as previously described. Pantosti et al., Infection and Immunity 59:2075-2082 (1991). The CPC from B. fragilis NCTC 9343 or ATCC 23745 was isolated by hot phenol/water extraction and subsequent purification of PSA and PSB performed as previously described. Tzianabos et al., The Journal of Biological Chemistry 267:18230-18235 (1992).

DEPR:
Inocula contained a 1:1 mixture of the challenge organism(s) and an adjuvant solution containing sterile rat cecal contents and 10% barium sulfate (w/v) as previously described. Onderdonk, A. et al., Infection and Immunity 13:22-26 (1976). Bacteria were grown anaerobically and, unless otherwise indicated, adjusted to the following concentrations (as determined by colony forming units on solid agar) per gelatin capsule: Bacteroides fragilis-5.times.10.sup.7, B. distasonis-5.times.10.sup.7, F. varium-2.5.times.10.sup.7, B. thetaiotaomicron-5.times.10.sup.7, and E. faecalis-1.3.times.10.sup.7. For some experiments, a cecal content inoculum containing feces procured from the ceca of meat-fed rats was used to challenge animals. Onderdonk, A. et al., Infection and Immunity 10:1256-1259 (1974). Quantitative and qualitative bacteriology, of the inoculum was performed and the results were as follows (CFU/ml -log.sub.10): Facultative organisms: Escherichia coli (6.32); group D Streptococcus (6.49); and alpha hemolytic Streptococcus (6.29); Obligate Anaerobes: Clostridium perfringes (8.05); Bacteroides thetaiotaomicron (6.90); Clostridium sordellii (6.70); Bacteroides vulgatus (6.70); Bacteroides caccae (6.30); and Bacteroides fragilis (6.00).

DEPR:
Following administration of 10 .mu.g doses of PSA according to Regimen 1, animals were challenged via the intraperitoneal route with various inocula containing bacteria commonly associated with abscesses found in humans. Groups of animals were challenged with a monomicrobial culture of B. fragilis, or mixed inocula consisting of combination of B. distasonis and E. faecalis, B. thetaiotaomicron and E. faecalis or F. varium and E. faecalis. In each case, fewer animals treated with PSA prior to bacterial challenge had abscesses than the saline-treated control group. (Table 8)

DEPR:
It was investigated whether cell-mediated immune mechanisms controlled the broadly protective activity exhibited by PSA in abrogating abscess formation by heterologous organisms commonly associated with intra-abdominal sepsis. Animals were treated with PSA (10 .mu.g/injection) according to Regimen 3 and T cells isolated 24 hours after last treatment. T cells (1.times.10.sup.7) from PSA-treated or saline-treated animals were administered to naive recipients 24 hours prior to challenge with B. fragilis or with a mixed inoculum of F. varium and E. faecalis. Results from these experiments are shown in Table 9. Animals receiving T cells from saline-treated rats and challenged with B. fragilis or the F. varium and E. faecalis combination developed abscesses (85% and 87%, respectively). However, transfer of T cells from PSA-treated animals to naive recipients yielded significant protection against abscess formation following challenge with B. fragilis (13% abscess rate) or F. varium and E. faecalis (21% abscess

DETL:
TABLE 8 _____ PSA-mediated protection against abscess formation. Abscess formation (No. Rats with Challenge abscesses/No. Treatment Inoculum Tested) P value _____ saline B. fragilis 15/18 (83%) -- saline B. distasonis + 16/18 (89%) -- E. faecalis saline B. thetaiotamicron + 15/19 (79%) -- E. faecalis saline F. varium + E. 13/15 (87%) -- faecalis PSA B. fragilis 1/19 (5%) <0.0001 PSA B. distasonis + 4/18 (22%) <0.001 E. faecalis PSA B. thetaiotaomicron + 2/19 (11%) <0.0001 E.

faecalis PSA F. varium + E. 5/17 (29%) <0.002 faecalis S. pneumoniae B. fragilis 9/10 (90%) NS type 3 CP

DETL:
TABLE 9 Abscess Formation (No. rats with Treatment Challenge abscesses/ (10 .mu.g) Inoculum No. tested) P value saline B. fragilis 11/13 (85%) -- saline F. varium and 13/15 (87%) -- E. faecalis PSA B. fragilis 1/8 (13%) <0.005 PSA F. varium and 3/14 (21%) <0.005 E. faecalis

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

☐ 3. Document ID: US 5019513 A

L8: Entry 3 of 3 File: USPT May 28, 1991

US-PAT-NO: 5019513
DOCUMENT-IDENTIFIER: US 5019513 A

TITLE: Anti-bacterial T-cell factor

DATE-ISSUED: May 28, 1991

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kasper; Dennis L.	Newton Center	MA		
Zaleznik; Dori F.	Newton Highlands	MA		
Finberg; Robert W.	Roslindale	MA		

US-CL-CURRENT: 435/340; 435/70.2

ABSTRACT:

A method of protecting a mammal against a pathogenic bacterium by administering a soluble suppressor T-cell factor derived from a mammal that has been immunized with the bacterium or an antigenic surface fragment of the bacterium. Also disclosed are a hybrid cell fusion of an immunized suppressor T cell, methods of making such cells, and method of producing soluble suppressor T-cell factors.

5 Claims, 0 Drawing figures Exemplary Claim Number: 1

L8: Entry 3 of 3 File: USPT May 28, 1991

DOCUMENT-IDENTIFIER: US 5019513 A
TITLE: Anti-bacterial T-cell factor

BSPR:
Ziegler et al. (1982) N.E. J. Med. 307:1225-1230 report treating human patients with human antiserum to bacterial endotoxin (lipopolysaccharide) prepared by vaccinating humans with heat killed Escherichia coli J5, a mutant having a core identical to most gram negative bacteria and lacking lipopolysaccharide oligosaccharide side chains.

DEPR:
ITF at concentrations of 2.5-25.times.10.sup.6 cell equivalents prevents the development of abcesses following challenge with viable B. fragilis to the same degree as 2.5.times.10.sup.6 intact immune T cells. No protection is provided against organisms such as B. distasonis (ATCC 8503) or Fusobacterium varium (TVDL 37). Neither 2.5.times.10.sup.6 nonimmune T cells or 25.times.10.sup.6 cell equivalents of NITF, a factor prepared from such T cells, provide any protection. Even a dose of 0.25.times.10.sup.6 cell equivalents of ITF provides a significant degree of protection compared to NITF. Crude factor prepared by lysing immune T cells leaving no cells intact by microscopic examination is as active as intact cells in preventing abscess formation in mice. Protective ITF is prepared from mouse splenic T cells at least 46 days following completion of the immunization protocol.

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☐ 1. Document ID: US 6107033 A

L16: Entry 1 of 1 File: USPT Aug 22, 2000

US-PAT-NO: 6107033
DOCUMENT-IDENTIFIER: US 6107033 A

TITLE: Methods and materials for determining relative abundance of microorganisms in mixed populations

DATE-ISSUED: August 22, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Welling; Gjalt Wietze	Roderwolde			NLX
Schut; Frederik	Groningen			NLX
Langendijk; Petra Simone	Nijmegen			NLX
Jansen; Gijsbert Johan	Groningen			NLX
Wilkingson; Michael Hendrik Francis	Groningen			NLX
Elffrich; Peter	Groningen			NLX

US-CL-CURRENT: 435/6; 435/5, 436/501, 536/23.1, 536/24.1, 536/24.3, 536/24.31, 536/24.32, 536/24.33

ABSTRACT:

The present invention relates to a method for determining the relative abundance of individual species or lower phylogenetic subgroups of microorganisms in a mixed population of several microorganisms comprising the steps of: 1) providing a set of labeled in situ hybridization cluster oligonucleotide probes; 2) hybridization of said probes with a sample of the mixed population, and 3) quantitative analysis of the number of labeled microorganisms. Further it relates to a method for analyzing dynamics in relative abundance of individual microorganisms in a mixed population. Further it relates to a set of probes which are cluster specific and which are provided with at least one label for use in a method of the invention and to a kit of parts for determining the relative abundance of individual species of microorganisms in a mixed population of several microorganisms comprising such probes which together with suitable materials for pre-treating the sample and/or suitable materials for carrying out hybridization and/or suitable materials for analysis of the result of the hybridization.

16 Claims, 1 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 4

L16: Entry 1 of 1 File: USPT Aug 22, 2000

DOCUMENT-IDENTIFIER: US 6107033 A
TITLE: Methods and materials for determining relative abundance of microorganisms in mixed populations

BSPW:
in combination with anti-DIG antibodies labelled with

BSPW:
in combination with avidin or streptavidin and labelled as the anti-DIG antibodies

BSPW:
in combinaiton with appropriate antibodies and labelled as the anti-DIG antibodies

DEPR:
5. The Fusobacterium genus probe Fus390 (CACACAGAATTGCTGGATC), Td=60.2.degree. C., has a a full match with Fusobacterium simiae, F. nucleatum, F. alocis, F. russii, F. gonidoformans, F. necrophorum, F. varium, F. mortiferum, F. perfoetens, Propionigenium modestum, Leptotrichia buccalis and Sebalcella termitidis. The probe may also hybridize to F. ulcerans, F. periodonticum and F. necrogenes since these bacteria have one unknown base in the target region.

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☐ 1. Document ID: US 6331550 B1

L23: Entry 1 of 1 File: USPT Dec 18, 2001

US-PAT-NO: 6331550
DOCUMENT-IDENTIFIER: US 6331550 B1

TITLE: Methods of use of quinolone compounds against anaerobic pathogenic bacteria

DATE-ISSUED: December 18, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Citron; Diane M.	Santa Monica	CA		
Goldstein; Ellie J. C.	Santa Monica	CA		

US-CL-CURRENT: 514/312; 514/300, 514/311

ABSTRACT:

This invention relates, in part, to newly identified methods of using quinolone antibiotics, particularly a gemifloxacin compound against pathogenic bacteria, especially anaerobic pathogens.

16 Claims, 0 Drawing figures Exemplary Claim Number: 1

L23: Entry 1 of 1 File: USPT Dec 18, 2001

DOCUMENT-IDENTIFIER: US 6331550 B1
TITLE: Methods of use of quinolone compounds against anaerobic pathogenic bacteria

BSPR:
A further object of the invention is a method wherein said anaerobic pathogenic bacteria is selected from the group consisting of: a member of the genus Peptostreptococci, a Peptostreptococci asaccharolyticus, a Peptostreptococci magnus, a Peptostreptococci micros, a Peptostreptococci prevotii, a Porphyromonas asaccharolytica, a Porphyromonas canoris, a Porphyromonas gingivalis, a Porphyromonas macaccae, a member of the genus Actinomyces, an Actinomyces israelii, an Actinomyces odontolyticus, a member of the genus Clostridium, a Clostridium innocuum, a Clostridium clostridioforme, a Clostridium difficile, a member of the genus Anaerobiospirillum, a Bacteroides tectum, a Bacteroides ureolyticus, a Bacteroides gracilis (Campylobacter gracilis), a Prevotella intermedia, a Prevotella heparinolytica, a Prevotella oris-buccae, a Prevotella bivia, a Prevotella melaninogenica, a member of the genus Fusobacterium, a Fusobacterium naviforme, a Fusobacterium necrophorum, a Fusobacterium varium, a Fusobacterium ulcerans, a Fusobacterium russii, a member of the genus Bilophila, a Bilophila wadsworthia.

BSPR:
Further preferred methods are provided by the invention wherein said bacteria is selected from the group consisting of: a member of the genus Peptostreptococci,

a Peptostreptococci asaccharolyticus, a Peptostreptococci magnus, a Peptostreptococci micros, a Peptostreptococci prevotii, a member of the genus Porphyromonas, a Porphyromonas asaccharolytica, a Porphyromonas canoris, a Porphyromonas gingivalis, a Porphyromonas macaccae, a member of the genus Actinomyces, an Actinomyces israelii, an Actinomyces odontolyticus, a member of the genus Clostridium, a Clostridium innocuum, a Clostridium clostridioforme, a Clostridium difficile, a member of the genus Anaerobiospirillum, a member of the genus Bacteroides, a Bacteroides tectum, a Bacteroides ureolyticus, a Bacteroides gracilis (Campylobacter gracilis), a member of the genus Prevotella, a Prevotelia intermedia, a Prevotella heparinolytica, a Prevotella oris-buccae, a Prevotella bivia, a Prevotella melaninogenica, a member of the genus Fusobacterium, a Fusobacterium naviforme, a Fusobacterium necrophorum, a Fusobacterium varium, a Fusobacterium ulcerans, a Fusobacterium russii, a member of the genus Bilophila, a Bilophila wadsworthia.

BSPR:
As provided herein, activities of gemifloxacin and comparator antimicrobial agents were determined by an agar dilution method against 419 clinical strains of less commonly identified, though medically important, species of anaerobes. Gemifloxacin was generally more active than trovafloxacin against Gram-positive strains by one to two dilutions. Peptostreptococci [P. asaccharolyticus, P. magnus, P. micros, and P. prevotii] and Porphyromonas spp. [P. asaccharolytica, P. canoris, P. gingivalis, P. macaccae] were all susceptible to {character pullout} 0.25 ug/ml of gemifloxacin. Actinomyces israelii, Actinomyces odontolyticus, Clostridium innocuum, Clostridium clostridioforme, Anaerobiospirillum spp., Bacteroides tectum, B. ureolyticus, B. gracilis [now Campylobacter gracilis], Prevotella intermedia, Prevotelia heparinolytica, Prevotella oris-buccae group had MIC.sub.90 s of {character pullout} 2 .mu.g/ml. Fusobacterium naviforme and F. necrophorum were also susceptible to {character pullout} 2 .mu.g/ml, while F. varium strains exhibited a bimodal pattern; the other Fusobacterium species, such as F. ulcerans, F. russii, as well as Veillonella spp., Prevotella melaninogenica group, P. bivia, Clostridium difficile, and Bilophila wadsworthia were relatively resistant to gemifloxacin [MIC.sub.90 s{character pullout} 4 .mu.g/ml).(See Table 1).

BSPR:
In addition to the therapy described above, a gemifloxacin compound or composition used in the methods of this invention may be used generally as a wound treatment agent to prevent adhesion of bacteria to matrix proteins, particularly anaerobic pathogenic bacteria, exposed in wound tissue and for prophylactic use in dental treatment as an alternative to, or in conjunction with, antibiotic prophylaxis.

BSPR:
While a preferred object of the invention provides a method wherein said anaerobic pathogenic bacteria is selected from the group consisting of: selected from the group consisting of: a member of the genus Peptostreptococci, a Peptostreptococci asaccharolyticus, a Peptostreptococci magnus, a Peptostreptococci micros, a Peptostreptococci prevotii, a member of the genus Porphyromonas, a Porphyromonas asaccharolytica, a Porphyromonas canoris, a Porphyromonas gingivalis, a Porphyromonas macaccae, a member of the genus Actinomyces, an Actinomyces israelii, an Actinomyces odontolyticus, a member of the genus Clostridium, a Clostridium innocuum, a Clostridium clostridioforme, a Clostridium difficile, a member of the genus Anaerobiospirillum, a member of the genus Bacteroides, a Bacteroides tectum, a Bacteroides ureolyticus, a Bacteroides gracilis (Campylobacter gracilis), a member of the genus Prevotella, a Prevotella intermedia, a Prevotella heparinolytica, a Prevotelia oris-buccae, a Prevotella bivia, a Prevotella melaninogenica, a member of the genus Fusobacterium, a Fusobacterium naviforme, a Fusobacterium necrophorum, a Fusobacterium varium, a Fusobacterium ulcerans, a Fusobacterium russii, a member of the genus Bilophila, a Bilophila wadsworthia.

BSTL:
TABLE 1 In vitro activity [ug/ml] gemifloxacin, trovafloxacin, and other oral antimicrobial agents against unusual anaerobic pathogens. Minimal Inhibitory

Concentration Organism & Agent (no. isolates).sup.A Range 50% 90% Actinomyces odontolyticus [10] Gemifloxacin 1-2 2 2 Trovafloxacin 2-4 4 4 Penicillin G 0.125-0.25 0.125 .0125 Amoxicillin clavulanate 0.06-0.125 0.125 0.25 Clindamycin {character pullout}0.015-0.5 0.125 0.25 Erythromycin {character pullout}0.015-0.03 {character pullout}0.015 0.03 Azithromycin {character pullout}0.015-0.06 0.03 0.06 Clarithromycin {character pullout}0.015 {character pullout}0.015 {character pullout}0.015 Metronidazole 4-> 32 16 32 Actinomyces israelii [6] Gemifloxacin 0.5-2 1 Trovafloxacin 0.5-2 1 Penicillin G {character pullout}0.015-0.25 0.03 Amoxicillin clavulanate 0.03-1 0.03 Clindamycin 0.06-0.25 0.06 Erythromycin 0.03 0.03 Azithromycin 0.06 0.06 Clarithromycin {character pullout}0.015 {character pullout}0.015 Metronidazole 1-32 4 Anaerobiospirillum thomasii [13] Gemifloxacin 0.06-0.25 0.125 0.125 Trovafloxacin 0.06-0.5 0.125 0.25 Penicillin G 0.06-0.125 0.06 0.125 Amoxicillin clavulanate 0.125-0.25 0.125 0.25 Clindamycin 8-> 32 32 >32 Erythromycin 1-16 4 8 Azithromycin 0.125-2 0.5 1 Clarithromycin 2-32 4 16 Metronidazole 1-4 2 4 Anaerobiospirillum succiniciproducens [33] Gemifloxacin 0.5-2 1 Trovafloxacin 0.5-2 1 Penicillin G 0.5-1 0.5 Amoxicillin clavulanate 0.25-0.5 0.25 Clindamycin 32 32 Erythromycin 8-16 16 Azithromycin 0.5-1 0.5 Clarithromycin 8-32 32 Metronidazole 4-8 8 Bacteroides gracilis [11] Gemifloxacin {character pullout}0.015-1 {character pullout}0.015 1 Trovofloxacin {character pullout}0.015-2 0.03 0.5 Penicillin G {character pullout}0.015-4 0.125 4 Amoxicillin clavulanate 0{character pullout}.015-2 0.5 2 Clindamycin 0.03-8 0.25 2 Erythromycin 0.125-2 1 2 Azithromycin 0.06-0.5 0.125 0.5 Clarithromycin 0.25-2 1 1 Metronidazole 0.06 > 32 0.5 >32 Bacteroides tectum [22] Gemifloxacin 0.06-8 0.125 0.25 Trovafloxacin 0.03-0.125 0.06 0.125 Penicillin G {character pullout}0.015-32 0.03 16 Amoxicillin clavulanate 0.03-0.5 0.06 0.5 Clindamycin {character pullout}0.015-0.125 {character pullout}0.015 {character pullout}0.015 Erythromycin 0.25-1 0.5 0.5 Azithromycin 0.5-2 1 2 Clarithromycin 0.125 0.125 0.125 Metronidazole 0.125-2 0.5 0.5 Bacteroides ureolyticus [17] Gemifloxacin {character pullout}0.015-2 {character pullout}0.015 2 Trovafloxacin {character pullout}0.015-4 0.06 4 Penicillin G {character pullout}0.015-1 {character pullout}0.015 0.25 Amoxicillin clavulanate {character pullout}0.015-1 {character pullout}0.015 0.125 Clindamycin 0.03-0.5 0.06 0.25 Erythromycin 0.125-2 .025 2 Azithromycin 0.06-0.25 0.06 0.25 Clarithromycin 0.125-4 0.5 2 Metronidazole 0.06-2 0.25 1 Bilophila wadsworthia [16] Gemifloxacin 0.125-> 8 0.25 4 Trovofloxacin 0.125-> 8 0.5 >8 Penicillin G 2-16 4 8 Amoxicillin clavulanate 1-4 2 4 Clindamycin 0.25-2 0.5 2 Erythromycin 4-32 16 32 Azithromycin 1-16 4 16 Clarithromycin 4-32 16 32 Metronidazole 0.125 0.125 0.125 Clostridium clostridioforme [11] Gemifloxacin 0.5-> 8 0.5 1 Trovafloxacin 1-8 4 4 Penicillin G 0.5-> 32 1 16 Amoxicillin clavulanate 0.5-8 0.5 1 Clindamycin {character pullout}0.015-2 0.06 2 Erythromycin 0.25-> 32 16 >32 Azithromycin 0.125-> 32 16 >32 Clarithromycin 0.125-> 32 4 >32 Metronidazole 0.03-1 0.125 0.5 Clostridium difficile [14] Gemifloxacin 1-> 8 2 >8 Trovafloxacin 0.5-> 8 1 >8 Penicillin G 1-4 2 4 Amoxicillin clavulanate 0.5-1 1 1 Clindamycin 0.25-> 32 0.5 >32 Erythromycin 0.25 > 32 0.5 >32 Azithromycin 1-> 32 2 >32 Clarithromycin 0.125-> 32 0.5 >32 Metronidazole 0.25-1 0.5 0.5 Clostridium inocuum [11] Gemifloxacin 0.125-> 8 0.25 2 Trovafloxacin 0.25-> 8 0.5 8 Penicillin G 0.25-> 3 0.5 0.5 Amoxicillin clavulanate 0.5-2 0.5 0.5 Clindamycin 0.25-> 32 0.5 >32 Erythromyccin 0.5-> 32 >32 >32 Azithromycin 0.125-> 32 >32 >32 Clarithromycin 0.25-> 32 >32 >32 Metronidazole 0.5-2 0.5 1 Clostridium ramosum [10] Gemifloxacin 0.125-2 0.25 1 Trovafloxcin 0.25-8 0.5 2 Penicillin G 0.06-1 0.06 1 Amoxicillin clavulanate 0.06-0.25 0.06 0.25 Clindamycin 0.25-4 2 2 Erythromycin 0.5-> 32 1 >32 Azithromycin 0.125-> 32 0.25 >32 Clarithromycin 0.25-> 32 0.5 >32 Metronidazole 1 1 1 Fusobacterium spp group 1 [19].sup.B Gemifloxacin 0.06-8 0.25 8 Trovafloxacin 0.25-4 0.5 4 Penicillin G {character pullout}0.015-16 {character pullout}0.015 2 Amoxicillin clavulanate {character pullout}0.015-0.25 0.06 0.125 Clindamycin {character pullout}0.015-2 0.06 0.125 Erythromycin 1-> 32 8 32 Azithromycin 0.06-32 1 8 Clarithromycin {character pullout}0.015-32 8 32 Metronidazole 0.125-0.5 0.25 4 Fusobacterium spp. group 2 [12].sup.C Gemifloxacin 0.125-> 8 4 4 Trovafloxacin 1-> 8 4 4 Penicillin G {character pullout}0.015-> 32 0.25 0.5 Amoxicillin clavulanate 0.125-> 4 1 2 Clindamycin 0.06-8 1 8 Erythromycin 8-> 32 >32 >32 Azithromycin 1-> 32 16 32 Clarithromycin 4-> 32 >32 >32 Metronidazole 0.125-1 0.5 1 Fusobacterium russii Gemifloxacin 0.5-> 8 >8 >8 Trovafloxacin 0.5-4 4 4 Penicillin G {character pullout}0.015-0.06 0.03 0.06 Amoxicillin clavulanate {character pullout}0.015-0.25 0.06 0.125

Clindamycin {character pullout}0.015-0.125 0.03 0.06 Erythromycin 1-> 32 4 >32 Azithromycin 0.03-32 0.25 32 Clarithromycin 2-> 32 4 >32 Metronidazole {character pullout}0.015-0.25 0.125 0.25 Fusobacterium varium [17] Gemifloxacin 0.25-> 8 >8 >8 Trovafloxacin 0.5-> 8 4 >8 Penicillin G 0.03-> 32 0.5 8 Amoxicillin clavulanate 0.125-4 2 4 Clindamycin 0.06-16 4 16 Erythromycin 32-> 32 >32 >32 Azithromycin 2-> 32 32 >32 Clarithromycin 32-> 32 >32 >32 Metronidazole 0.125-4 1 2 Peptostreptococcus asaccharolyticus [11] Gemifloxacin 0.125-0.25 0.25 0.25 Trovafloxacin 0.5-2 1 1 Penicillin G {character pullout}0.015-1 0.03 0.25 Amoxicillin clavulanate 0.03-1 0.03 0.125 Clindamycin {character pullout}0.015-> 32 0.06 >32 Erythromycin 1-> 32 4 >32 Azithromycin 0.5-> 32 4 >32 Clarithromycin 0.5-> 32 2 >32 Metronidazole 0.125-2 0.5 1 Peptostreptococcus magnus [13] Gemifloxacin 0.030.03 0.03 0.06 Trovafloxacin 0.06-0.25 0.125 0.25 Penicillin G {character pullout}0.015-1 0.03 0.25 Amoxicillin clavulanate 0.03-1 0.03 0.125 Clindamycin 0.06-2 0.5 2 Erythromycin 1-> 32 4 >32 Azithromycin 2-> 32 4 >32 Clarithromycin 0.5-> 32 2 >32 Metronidazole 0.25-2 0.5 0.5 Peptostreptoeoccus micros [12] Gemifloxacin 0.06-0.125 0.06 0.06 Trovafloxacin 0.03-0.125 0.06 0.06 Penicillin G {character pullout}0.015-0.03 {character pullout}0.015 0.03 Amoxicillin clavulanate 0.03-0.125 0.03 0.125 Erythromycin 0.5-1 0.5 0.5 Azithromycin 0.5-1 0.5 1 Clarithromycin 0.6 0.5 0.5 Clindamycin 0.06-0.125 0.125 0.125 Metronidazole 0.03-0.25 0.25 0.25 Peptostreptococcus prevotii [9] Gemifloxacin 0.06-0.25 0.125 -- Trovafloxacin 0.25-1 0.25 -- Penicillin G 0.03-0.06 0.03 -- Amoxicillin clavulanate {character pullout}0.015-0.125 0.03 -- Clindamycin 0.030-32 1 -- Erythromycin 0.03-> 32 >32 -- Azithromycin 0.06-> 32 32 -- Clarithromycin {character pullout}0.015-> 32 >32 -- Metronidazole 0.125-1 0.5 -- Porphyromonas asaccharolyticus [11] Gemifloxacin 0.06-0.125 0.06 0.125 Trovafloxacin 0.03-0.25 0.25 0.25 Penicillin G {character pullout}0.015 {character pullout}0.015 {character pullout}0.015 Amoxicillin clavulanate {character pullout}0.015-0.03 {character pullout}0.015 0.03 Clindamycin {character pullout}0.015-> 32 {character pullout}0.015 >32 Erythromycin 0.03-32 0.03 32 Azithromycin 0.125-> 32 0.25 >32 Clarithromycin {character pullout}0.015-> 32 0.06 >32 Metronidazole {character pullout}0.015 {character pullout}0.015 {character pullout}0.015 Porphyromonas canoris [10] Gemifloxacin 0.06-0.25 0.25 0.25 Trovafloxacin 0.06-0.5 0.25 0.5 Penicillin G {character pullout}0.015-0.03 {character pullout}0.015 {character pullout}0.015 Amoxicillin clavulanate {character pullout}0.015-0.03 {character pullout}0.015 0.03

CLPV:
Peptostreptococci asaccharolyticus, Peptostreptococci magnus, Peptostreptococci micros, Peptostreptococci prevotii, Porphyromonas asaccharolytica, a Porphyromonas canoris, Porphyromonas gingivalis, Porphyromonas macaccae, Actinomyces israelii, Actinomyces odontolyticus, Clostridium innocuum, Clostridium clostridioforme, Clostridium difficile, Bacteroides tectum, Bacteroides ureolyticus, Bacteroides gracilis (Campylobacter gracilis), Prevotella intermedia, Prevotella heparinolytica, Prevotella oris-buccae, Prevotella bivia, Prevotella melaninogenica, Fusobacterium naviforme, Fusobacterium necrophorum, Fusobacterium varium, Fusobacterium ulcerans, Fusobacterium russii, and Bilophila wadsworthia.

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☐ 1. Document ID: US 6162927 A

L33: Entry 1 of 8 File: USPT Dec 19, 2000

US-PAT-NO: 6162927
DOCUMENT-IDENTIFIER: US 6162927 A

TITLE: Endothelin antagonists

DATE-ISSUED: December 19, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
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von Geldern; Thomas W.	Richmond	IL		
Kester; Jeffrey A.	Deerfield	IL		
Sorensen; Bryan K.	Waukegan	IL		
Szczepankiewicz; Bruce G.	Gages Lake	IL		
Henry; Kenneth J.	Waukegan	IL		
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US-CL-CURRENT: 548/526; 548/517, 548/518, 548/525, 548/531, 548/541

ABSTRACT:

A compound of the formula (I): ##STR1## or a pharmaceutically acceptable salt thereof is disclosed, as well as processes for and intermediates in the preparation thereof, and a method of antagonizing endothelin.

10 Claims, 0 Drawing figures Exemplary Claim Number: 2,3

L33: Entry 1 of 8 File: USPT Dec 19, 2000

DOCUMENT-IDENTIFIER: US 6162927 A
TITLE: Endothelin antagonists

BSPR:
Agents with the ability to antagonize ET/ET receptor binding have been shown to be active in a number of animal models of human disease. For example, Hogaboam et al (EUR. J. Pharmacol. 1996, 309, 261-269), have shown that an endothelin receptor antagonist reduced injury in a rat model of colitis. Aktan et al (Transplant Int 1996, 9, 201-207) have demonstrated that a similar agent prevents ischemia-reperfusion injury in kidney transplantation. Similar studies have suggested the use of endothelin antagonists in the treatment of angina, pulmonary hypertension, Raynaud's disease, and migraine. (Ferro and Webb, Drugs 1996, 51,12-27).

DEPR:
Ethyl 2-(4-methoxybenzoyl)-4-nitromethyl-3-(1,3-benzodioxol-5-yl) butyrate

DEPR:
The compounds of the present invention can be used in the form of salts derived from inorganic or organic acids. These salts include but are not limited to the following: acetate, adipate, alginate, citrate, aspartate, benzoate, benzenesulfonate, bisulfate, butyrate, camphorate, camphorsulfonate, digluconate, cyclopentanepropionate, dodecylsulfate, ethanesulfonate, glucoheptanoate, glycerophosphate, hemisulfate, heptanoate, hexanoate, fumarate, hydrochloride, hydrobromide, hydroiodide, 2-hydroxy-ethanesulfonate, lactate, maleate, methanesulfonate, nicotinate, 2-naphthalenesulfonate, oxalate, pamoate, pectinate, persulfate, 3-phenylpropionate, picrate, pivalate, propionate, succinate, tartrate, thiocyanate, p-toluenesulfonate and undecanoate. Also, the basic nitrogen-containing groups can be quaternized with such agents as loweralkyl halides, such as methyl, ethyl, propyl, and butyl chloride, bromides, and iodides; dialkyl sulfates like dimethyl, diethyl, dibutyl, and diamyl sulfates, long chain halides such as decyl, lauryl, myristyl and stearyl chlorides, bromides and iodides, aralkyl halides like benzyl and phenethyl bromides, and others. Water or oil-soluble or dispersible products are thereby obtained.

DEPC:
Ethyl 2-(4-methoxybenzoyl)-4-nitromethyl-3-(1,3-benzodioxole-5-yl)butyrate

DEPC:
Alternate Prepration of Ethyl
2-(4-methoxybenzoyl)-4-nitromethyl-3-(1,3-benzodioxole-5-yl)butyrate

DEPC:
Ethyl 2-(4-methoxyphenyl)oxo-4-nitro-3-(3,4-methylenedioxyphenyl)butyrate

DEPC:
Methyl 2-(4-hexenoyl)-4-nitro-3-(1,3-benzodioxole-5-yl)butyrate

DEPC:
Ethyl 2-(4-butanoyl)-4-nitro-3-(1,3-benzodioxole-5-yl)butyrate

Full | Title | Citation | Front | Review | Classification | Date | Reference
KWIC | Draw Desc | Image

☐ 2. Document ID: US 6110922 A

L33: Entry 2 of 8 File: USPT Aug 29, 2000

US-PAT-NO: 6110922
DOCUMENT-IDENTIFIER: US 6110922 A

TITLE: Cell adhesion-inhibiting antiinflammatory and immune-suppressive compounds

DATE-ISSUED: August 29, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Link; James	Evanston	IL		
Liu; Gang	Gurnee	IL		
Pei; Zhonghua	Libertyville	IL		
Geldern; Tom von	Richmond	IL		
Winn; Martin	Deerfield	IL		
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US-CL-CURRENT: 514/259; 514/395, 514/415, 514/712, 544/253, 544/282, 548/306.4, 549/362, 549/469, 568/58

ABSTRACT:

The present invention relates to novel cinnamide compounds that are useful for treating inflammatory and immune diseases, to pharmaceutical compositions comprising these compounds, and to methods of inhibiting inflammation or suppressing immune response in a mammal.

19 Claims, 0 Drawing figures Exemplary Claim Number: 1

L33: Entry 2 of 8 File: USPT Aug 29, 2000

DOCUMENT-IDENTIFIER: US 6110922 A
TITLE: Cell adhesion-inhibiting antiinflammatory and immune-suppressive compounds

BSPR:
The compounds of the present invention may be used in the form of pharmaceutically-acceptable salts derived from inorganic or organic acids. By "pharmaceutically-acceptable salt" is meant those salts which are, within the scope of sound medical judgment, suitable for use in contact with the tissues of humans and lower animals without undue toxicity, irritation, allergic response and the like, and are commensurate with a reasonable benefit/risk ratio. Pharmaceutically-acceptable salts are well-known in the art. For example, S. M. Berge, et al. Describe pharmaceutically-acceptable salts in detail in J. Pharmaceutical Sciences, 1977, 66: 1 et seq. The salts may be prepared in situ during the final isolation and purification of the compounds of the invention or separately by reacting a free base function with a suitable acid. Representative acid addition salts include, but are not limited to acetate, adipate, alginate, citrate, aspartate, benzoate, benzenesulfonate, bisulfate, butyrate, camphorate, camphorsulfonate, digluconate, glycerophosphate, hemisulfate, heptanoate, hexanoate, fumarate, hydrochloride, hydrobromide, hydroiodide, 2-hydroxyethanesulfonate (isethionate), lactate, maleate, methanesulfonate, nicotinate, 2-naphthalenesulfonate, oxalate, pamoate, pectinate, persulfate, 3-phenylpropionate, picrate, pivalate, propionate, succinate, tartrate, thiocyanate, phosphate, glutamate, bicarbonate, p-toluenesulfonate and undecanoate. Also, the basic nitrogen-containing groups can be quaternized with such agents as lower alkyl halides such as methyl, ethyl, propyl, and butyl chlorides, bromides and iodides; dialkyl sulfates like dimethyl, diethyl, dibutyl and diamyl sulfates; long chain halides such as decyl, lauryl, myristyl and stearyl chlorides, bromides and iodides; arylalkyl halides like benzyl and phenethyl bromides and others. Water or oil-soluble or dispersible products are thereby obtained. Examples of acids which may be employed to form pharmaceutically acceptable acid addition salts include such inorganic acids as

hydrochloric acid, hydrobromic acid, sulphuric acid and phosphoric acid and such organic acids as oxalic acid, maleic acid, succinic acid and citric acid.

DEPR:
The ability of compounds of this invention to treat inflammatory bowel disease can be demonstrated in a rabbit chemical-induced colitis model according to the method of Bennet et al., J Pharmacol Exp Ther 280:988-1000, 1997.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw. Desc | Image

☐ 3. Document ID: US 5968894 A

L33: Entry 3 of 8 File: USPT Oct 19, 1999

US-PAT-NO: 5968894
DOCUMENT-IDENTIFIER: US 5968894 A

TITLE: Verotoxin pharmaceutical compositions and medical treatments therewith

DATE-ISSUED: October 19, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lingwood; Clifford A.	Toronto			CAX
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US-CL-CURRENT: 514/2

ABSTRACT:

Pharmaceutical compositions comprising known verotoxins, particularly, verotoxin 1 and their pentameric subunit B, have been found to be useful in the treatment of mammalian neoplasia, particularly, brain cancer, ovarian cancer, breast cancer and skin cancer. Although verotoxin 1 has previously been shown to have anti-neoplastic activity in vitro, non-lethal doses of verotoxin 1 have been shown to be therapeutically anti-neoplastic in vivo. Use of a sensitizer, such as sodium butyrate, enhances the efficacy of verotoxins and their subunit B.

12 Claims, 24 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 14

L33: Entry 3 of 8 File: USPT Oct 19, 1999

DOCUMENT-IDENTIFIER: US 5968894 A
TITLE: Verotoxin pharmaceutical compositions and medical treatments therewith

ABPL:
Pharmaceutical compositions comprising known verotoxins, particularly, verotoxin 1 and their pentameric subunit B, have been found to be useful in the treatment of mammalian neoplasia, particularly, brain cancer, ovarian cancer, breast cancer and skin cancer. Although verotoxin 1 has previously been shown to have anti-neoplastic activity in vitro, non-lethal doses of verotoxin 1 have been shown to be therapeutically anti-neoplastic in vivo. Use of a sensitizer, such as sodium butyrate, enhances the efficacy of verotoxins and their subunit B.

BSPR:

The verotoxin family of E coli elaborated toxins bind to the globo series glycolipid globotriaosylceramide and require terminal gal .alpha.-1-4 gal residue for binding. In addition, VT2e, the pig edema disease toxin, recognizes globotetraosylceramide (Gb.sub.4) containing an additional .beta. 1-3 linked galNac residue. These glycolipids are the functional receptors for these toxins since incorporation of the glycolipid into receptor negative cells renders the recipient cells sensitive to cytotoxicity. The toxins inhibit protein synthesis via the A subunit--an N-glycanase which removes a specific adenine base in the 28S RNA of the 60S RNA ribosomal subunit. However, the specific cytotoxicity and specific activity is a function of the B subunit. In an in vitro translation system, the verotoxin A subunit is the most potent inhibitor of protein synthesis yet described, being effective at a concentration of about 8 pM. In the rabbit model of verocytotoxemia, pathology and toxin targeting is restricted to tissues which contain the glycolipid receptor and these comprise endothelial cells of a subset of the blood vasculature. Verotoxins have been strongly implicated as the etiological agents for hemolytic uremic syndrome and haemorrhagic colitis, microangiopathies of the glomerular or gastrointestinal capillaries respectively. Human umbilical vein endothelial cells (HUVEC) are sensitive to verotoxin but this sensitivity is variable according to cell line. Human adult renal endothelial cells are exquisitely sensitive to verotoxin in vitro and express a correspondingly high level of Gb.sub.3. However, HUS is primarily a disease of children under three and the elderly, following gastrointestinal VTEC infection. It has been shown that receptors for verotoxin are present in the glomeruli of infants under this age but are not expressed in the glomeruli of normal adults. HUVEC can be sensitized to the effect of verotoxin by pretreatment by tumour necrosis factor which results in a specific elevation of Gb.sub.3 synthesis (7,8). Human renal endothelial cells on the other hand, although they express high levels of Gb.sub.3 in culture, cannot be stimulated to increase Gb.sub.3 synthesis (8). It has been suggested that the transition from renal tissue to primary endothelial cell culture in vitro results in the maximum stimulation of Gb.sub.3 synthesis from a zero background (9). We therefore suspect that HUS in the elderly is the result of verotoxemia and a concomitant stimulation of renal endothelial cell Gb.sub.3 synthesis by some other factor, eg. LPS stimulation of serum .alpha. TNF. Thus under these conditions, the majority of individuals (excepting the very young) would not be liable to VT induced renal pathology following systemic verotoxemia.

BSPR:
A series of human Gb.sub.3 containing astrocytoma cell lines were tested for sensitivity to VT. Although all cells were sensitive, the sensitivity varied over a 5000-fold range despite approximately equivalent Gb.sub.3 levels. We have found that treatment of the least sensitive cell line with sodium butyrate initiated a 5000-fold increase in VT sensitivity concomitant with an alteration in intracellular VT targeting.

BSPR:
Thus, we have also found that the efficacy of verotoxin and its B subunit may be significantly enhanced by a prior treatment of the neoplastic cells with a sensitizer, such as sodium butyrate.

DEPR:
FIG. 13 compares the sensitivity of two astrocytoma cell lines SF539 (sensitive), XF498 (less sensitive) and XF 498, following three days of culture of XF498 in sodium butyrate. It is seen that the sensitivity of XF498 is increased to that or even more than that of the most sensitive cell line SF539. FIG. 14 shows the same effect for the B subunit of verotoxin 1.

CLPR:
4. The method of claim 3, wherein said sensitizer is sodium butyrate.

CLPR:
9. The method of claim 8, wherein said sensitizer is sodium butyrate.

☐ 4. Document ID: US 5952314 A

L33: Entry 4 of 8 File: USPT Sep 14, 1999

US-PAT-NO: 5952314
DOCUMENT-IDENTIFIER: US 5952314 A

TITLE: Nutritional product for a person having ulcerative colitis

DATE-ISSUED: September 14, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
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Garleb; Keith Allen	Powell	OH	43081	
McEwen; John William	Gahanna	OH	43230	
Fuller; Martha Kay	Westerville	OH	43081-3602	

US-CL-CURRENT: 514/54; 426/567, 426/658, 514/168, 514/188, 514/552, 514/566, 514/725, 514/810, 514/812, 514/813, 514/861

ABSTRACT:

An enteral nutritional product for a person having ulcerative colitis contains in combination (a) an oil blend which contains eicosapentaenoic acid (20:5n3) and/or docosahexaenoic acid (22:6n3), and (b) a source of indigestible carbohydrate which is metabolized to short chain fatty acids by microorganisms present in the human colon. Preferably the nutritional product also contains one or more nutrients which act as antioxidants.

16 Claims, 5 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 5

L33: Entry 4 of 8 File: USPT Sep 14, 1999

DOCUMENT-IDENTIFIER: US 5952314 A
TITLE: Nutritional product for a person having ulcerative colitis

DEPR:
Increasing interest has been generated in the use of enemas/irrigation solutions containing buffered, physiologic levels of SCFAs for the treatment of diversion colitis and ulcerative colitis. Diversion colitis is an inflammatory process arising in segments of the colorectum at various intervals after surgical diversion of the fecal stream. The endoscopic appearance is similar to those of active Crohn's Disease and ulcerative colitis. Glotzer et al., "Proctitis and Colitis Following Diversion of the Fecal Stream", GASTROENTEROLOGY Vol. 80, pages 438-441 (1981). The cause of this condition is not known, but one mechanism has been postulated; a nutritional deficiency of the colonic epithelium, specifically due to the absence of SCFAs normally present in colonic contents, Komorowski, "Histologic Spectrum of Diversion Colitis" AMERICAN JOURNAL OF SURGICAL PATHOLOGY, Vol. 14, page 548 (1990), Roediger, "The Starved Colon--Diminished Mucosal Nutrition, Diminished Absorption, and Colitis", DISEASES OF THE COLON AND RECTUM, Vol. 33, pages 858-862 (1990). Harig et al., "Treatment of Diversion Colitis with Short-Chain-Fatty Acid Irrigation", NEW ENGLAND JOURNAL OF MEDICINE, Vol. 310, pages 23-28 (1989) tested this hypothesis by assessing whether irrigation with SCFAs could ameliorate inflammation in four patients with diversion colitis. These patients were administered SCFAs twice daily for 2-3 weeks with 60 mL of an enema solution comprising a physiologic mixture of SCFAs as sodium salts. After 2-3 weeks of therapy, macroscopic and histological resolution of inflammation was evident. An impaired utilization of

SCFAs has also been implicated in ulcerative colitis which suggests that diminished intracellular energy production may be important in the inflammatory process, Roediger, "The Colonic Epithelium in Ulcerative Colitis: an Energy Deficiency Disease?", THE LANCET, Vol. 2, pages 712-715 (1980). Vernia et al., "Fecal Lactate and Ulcerative Colitis", GASTROENTEROLOGY, Vol. 95, pages 1564-1568 (1988); and Vernia et al., "Organic Anions and the Diarrhea of Inflammatory Bowel Disease", DIGESTIVE DISEASES AND SCIENCES, Vol. 33, pages 1353-1358 (1988) have shown that fecal water from patients with ulcerative colitis contains reduced concentrations of SCFAs as well as markedly increased lactate and low pH. In a study by Breuer et al., "Rectal Irrigation with Short-Chain Fatty Acids for Distal Ulcerative Colitis" (preliminary report), DIGESTIVE DISEASES AND SCIENCES, Vol. 36, pages 185-187 (1991), relates an investigation of large bowel irrigation with SCFAs in patients with ulcerative colitis. It was found that 9 out of 10 patients completing the study were judged to be at least much improved and showed a significant change in mean disease activity index score and mucosal histology score. Recently Senagore et al., "Short-Chain Fatty Acid Enemas: a Cost Effective Alternative in the Treatment of Nonspecific Proctosigmoiditis", DISEASES OF THE COLON AND RECTUM, Vol. 35, page 923 (1992), confirmed the results of Breuer et al. demonstrating an 80 percent response rate in patients with idiopathic proctosigmoiditis. This study indicates that administering a solution of SCFAs similar to Harig et al. for six weeks was equally efficacious to corticosteroid or 5-ASA enemas for the treatment of proctosigmoiditis at a significant cost savings. Scheppach et al., "Effect of Butyrate Enemas on the Colonic Mucosa in Distal Ulcerative Colitis", GASTROENTEROLOGY, Vol. 103, pages 51-56 (1992) investigated the use of butyrate enemas alone rather than the SCFA mixture to treat ten patients with distal ulcerative colitis in a placebo-controlled, single-blind, randomized trial. The authors concluded that markedly improved disease activity index and histological parameters suggesting that the effect of a SCFA mixture on the inflamed mucosa in ulcerative colitis is largely attributable to its butyrate moiety.

DEPR:
Certain of the organisms that inhabit the large bowel can utilize dietary fiber (eg, pectin and gum arabic) and indigestible oligosaccharides (eg, fructooligosaccharides and xylooligosaccharides) as an energy source. Smith et al., "Introduction to Metabolic Activities of Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 32, pages 149-157 (1979); Miller et al., "Fermentation by Saccharolytic Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 32, pages 164-172 (1979); Cummings., "Fermentation in the Human Large Intestine: Evidence and Implications for Health", THE LANCET, Vol. 1, pages 1206-1209 (1983); Titgemeyer et al., "Fermentability of Various Fiber Sources by Human Fecal Bacteria In Vitro", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 53, pages 1418-1424 (1991). The microorganisms derive energy from the carbohydrate sources through a process referred to as anaerobic fermentation. During fermentation, the microorganisms produce SCFAs (eg, acetate, propionate, butyrate) as the major end products. Salyers et al., "Fermentation of Mucin and Plant Polysaccharides by Strains of Bacteroides from the Human Colon", APPLIED AND ENVIRONMENTAL MICROBIOLOGY, Vol. 33, pages 319-322 (1977); Mitsuoka et al., "Effect of Fructo-oligosaccharides on Intestinal Microflora", DIE NAHRUNG, Vol. 31, pages 427-436 (1987); Tokunaga et al., "Influence of Chronic Intake of a New Sweetener Fructooligosaccharide (Neosugar) on growth and Gastrointestinal Function of the Rat", JOURNAL OF NUTRITIONAL SCIENCE AND VITAMINOLOGY, Vol. 32, pages 111-121 (1986).

DEPR:
Analysis of acetate, propionate and butyrate was conducted according to Merchen et al., "Effect of Intake and Forage Level on Ruminal and Turnover Rates, Bacterial Protein Synthesis and Duodenal Amino Acid Flows in Sheep", JOURNAL OF ANIMAL SCIENCE, Vol. 62, pages 216-225 (1986). Briefly, an aliquot from the balch tube was acidified with 6N HCl and centrifuged at 31,000.times.g for 20 minutes Concentrations of acetate, propionate and butyrate were-determined in the supernatant using a Hewlett-Packard 5890A gas chromatograph and a column (180 cm.times.4 mm id) packed with 20% Tween 80-2% H.sub.3 PO.sub.4 on 60 to 80 mesh Chromosorb W (Supelco Inc, Bellefonte, Pa., U.S.A.). Nitrogen was used as a carrier gas with a flow rate of 70 mL/minutes. Oven temperature was 120.degree.

C. and detector and injector temperatures were 200.degree. C. Lactate was determined colorimetrically using a method described in Barker et al., "The Colorimetric Determination of Lactic Acid in Biological Material", JOURNAL OF BIOLOGICAL CHEMISTRY, Vol. 138, page 535, (1941).

DEPR:
SCFA production (acetate, propionate, butyrate and lactate) during in vitro fermentation of the oligosaccharides is presented in Table 3. Four time points were studied and include 3, 6, 12 and 24 hours. Retention time in the large bowel of humans will dictate the length of fermentation in vivo. In cases where retention time is great, the extent of substrate fermentability will be a factor which most influences SCFA production. If retention time is short, the rate of substrate fermentation becomes more important. Since retention times can differ significantly in an in viva situation it is necessary to monitor substrate degradation over time in vitro in order that comparisons can be made.

DEPR:
Fermentation of all oligosaccharides was rapid, essentially being complete by 6 hours for the fructooligosaccharides (FOS and Raftilose) and by 12 hours for XOS. The results are presented in Table 5. It is recommended that the 6 hour and 12 hour values be used to estimate the composition of the end-products for the fructooligosaccharides and the XOS, respectively, even though retention times in the large bowel can be considerably longer. At later time points it becomes apparent that lactate is being converted to propionate and acetate to butyrate. Interconversion in a closed in vitro system can be a problem with rapidly fermented substrates. It does not reflect the true state of the large bowel where the fatty acids are continually absorbed.

DEPR:
As is typically found with in vitro fermentations using human fecal inoculum or in analysis of fecal samples, acetate was the short chain fatty acid found in the highest concentration. Titgemeyer et al., "Fermentability of Various Fiber Sources by Human Fecal Bacteria In Vitro", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 53, Pages 1418-1424, (1991). Baldwin., "Energy Metabolism in Anaerobes", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 23, pages 1508-1513, (1970). Determined that acetate, propionate and butyrate account for 83% of the SCFAs produced during anaerobic fermentation by large bowel microflora, and the remaining SCFAs are distributed among isovaleric, isobutyric, valeric, lactic, formic and succinic acids. In this study, a considerable amount of lactate was found, particularly during fermentation with FOS and Raftilose. It has been documented that the oligosaccharides used in this study serve as an energy source for Bifidobacteria and that there consumption will lead to the selective growth of this organism in the GI tract. Okazaki et al., "Effects of Xylooligosaccharides on the Growth of Bifidobacteria", BIFIDOBACTERIA MICROFLORA, Vol. 9, page 77, (1990); Mitsuoka et al., "Effects of Fructo-oligosaccharide on Intestinal Microflora", DIE NAHRUNG, Vol. 31, pages 427-436, (1987). The primary end products produced by Bifidobacteria during fermentation are acetate and lactate. Miller et al., "Fermentations by Saccharolytic Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 32, pages 164-172, (1979). The fact that these oligosaccharides serve as an energy source for the Bifidobacteria could explain the elevated levels of lactate found in this study.

DEPR:
Total short-chain fatty acid production was greater for the xylooligosaccharides (XOS) compared to the fructooligosaccharides (FOS and Raftilose). The primary factor effecting the quantity of SCFAs produced during fermentation is the fermentability of the substrate. It is assumed that the oligosaccharides are completely fermented in this system. However, the yield of SCFAs (mol) from a substrate is dependent not only on the weight of the substrate fermented but also on the average molecular weight of the oligosaccharide component sugars. One can assume that the fermentation of one monosaccharide molecule can result in either two acetate, two propionate, two lactate or one molecule of butyrate. The molecular weight of the components of the fructooligosaccharides (glucose and fructose, 180) is greater than the molecular weight of xylose (150) which is

the monomeric component of XOS. Subsequently, on an equivalent weight basis, there are more moles of monosaccharide molecules with the xylooligosaccharide compared to the fructooligosaccharide. This would explain the greater production of SCFA with the XOS compared to the fructooligosaccharides. Lastly, the quantity and profile of SCFAs produced was virtually identical between the two fructooligosaccharides (Raftilose and FOS). While these fructooligosaccharides differ to some extent in their chemical composition, it is apparent that they are metabolized similarly in this in vitro fermentation system.

DEPR:
Recent evidence that the regular intake of n-3 fatty acids from fish oil inhibits neutrophil and monocyte functions suggests that n-3 fatty acids have antiinflammatory properties. Beneficial effects of marine lipids have been shown in animal models of inflammatory bowel disease. Empey et al., "Fish Oil-Enriched Diet is Mucosal Protective Against Acetic Acid-Induced Colitis in Rats", CANADIAN JOURNAL OF PHYSIOLOGY AND PHARMACOLOGY, Vol. 69, pages 480-487 (1991); Vilaseca et al., "Dietary Fish Oil Reduces Progression of Chronic Inflammatory Lesions in a Rat Model of Granulomatous Colitis", GUT, Vol. 31, pages 539 (1990). In preliminary therapeutic trials, diet supplementation with fish oil has led to symptomatic improvement of patients with ulcerative colitis, and reduced ethanol-induced damage in human duodenal mucosa. Schepp et al., "Fish Oil Reduces Ethanol-Induced Damage of the Duodenal Mucosa in Humans", GASTROENTEROLOGY, Vol. 96, page 446 (1989); Lorenz et al., "Supplementation with n-3 Fatty Acids from Fish Oil in Chronic Inflammatory Bowel Disease", JOURNAL OF INTERNAL MEDICINE SUPPLEMENT, Vol. 225, pages 225-232 (1989); Hillier et al., "Incorporation of Fatty Acids from Fish Oiliand Olive Oil into Colonic Mucosal Lipids and Effects Upon Eicosanoid Synthesis in Inflammatory Bowel Disease", GUT, Vol. 32, pages 1151-1155 (1991); Saloman et al., "Treatment of Ulcerative Colitis with Fish Oil N-3-w-Fatty Acid: An Open Trial", JOURNAL OF CLINICAL GASTROENTEROLOGY, Vol. 12, No. 2, pages 157-161 (1990).

DEPR:
A major limitation in investigating the pathogenic mechanisms responsible for the mucosal injury observed during chronic inflammation of the intestine and colon has been the relative paucity of relevant animal models. Two models of colitis produced in rats that have received much attention over the past few years are the acetic acid and trinitrobenzene sulfonic acid (TNBS) models. The mechanism by which acetic acid produces the diffuse colitis is thought to involve nonspecific, acid induced injury to the colonic mucosa that is followed by an acute inflammatory response. Apparently the protonated form of the acid is required to induce the colitis since neither HCl (pH 2.3) nor sodium acetate (pH 7.0) is effective in eliciting the inflammatory response. However, there is some evidence to suggest that acetic acid may promote other pathophysiological events (e.g. fluid and electrolyte secretion) using noncytotoxic concentrations of the acid.

DEPR:
Recent studies have demonstrated that the intrarectal administration of the hapten, TNBS, in the presence of a mucosal barrier breaker such as ethanol, produces an acute and possibly chronic colitis in unsensitized rats. The mechanism(s) by which buffered or unbuffered TNBS in the presence of ethanol initiates inflammation in unsensitized animals is unclear; however, it has been suggested to involve macrophage-mediated recognition and lysis of TNBS-modified autologous cells within the mucosa. However, more recent evidence suggests more complicated mechanisms. For example, the barrier breaker, ethanol, is an extremely potent pro-inflammatory solvent alone. Furthermore, it has been demonstrated that TNBS is metabolized by certain colonic enzymes and substrates to yield both pro-inflammatory and cytotoxic oxidants that could initiate colonic inflammation. Grisham et al., "Metabolism of Trinitrobenzene Sulfonic Acid by the Rat Colon Produces Reactive Oxygen Species", GASTROENTEROLOGY, Vol. 101, pages 540-547 (1991). A recent study directly compared the acetic acid and the TNBS (+ETOH) models of colitis and found that either model may be useful to study those events that occur at the time of inflammation (e.g. arachidonate metabolism, granulocyte infiltration and metabolism, etc.) or during repair. However, the use of these models of colitis may have significant limitations in

understanding those immunological events that initiate the acute and chronic inflammatory episodes. For example, the inflammation and tissue injury observed in human inflammatory bowel disease is most probably a result of inappropriate immunological activation (e.g. autoimmune, infectious agent, etc.) whereas the inflammation induced by the intrarectal application of acetic acid, ethanol or ethanol plus TNBS is a response to extensive mucosal injury. Thus, the mechanisms by which inflammation (and mucosal injury) are achieved in the human disease may be very different than those in the experimental models.

DEPR:
For these reasons, a model of acute and chronic distal colitis in rats was developed based upon a previously published method in which purified bacterial cell wall polymers (derived from Group A streptococci) are injected intramurally into the distal colon of genetically-susceptible rats. Sartor et al., "Granulomatus Entercolitis Induced by Purified Bacterial Cell Wall Fragments", GASTROENTEROLOGY, Vol. 89, pages 587-595 (1985). This model produces an acute and chronic inflammation characterized by the infiltration of large numbers of inflammatory cells, enhanced mucosal permeability, interstitial fibrosis, and mucosal thickening as well as the extraintestinal manifestations of arthritis, hepatic and splenic granulomas. Unlike most models of colitis, the inflammation induced in this model promoting mucosal and submucosal injury rather than the injury causing the inflammation.

DEPR:
In Experiment 2 the objectives were: (a) to determine whether this model of colitis responds to sulfasalazine (SAZ) and (b) to assess the effects of specially formulated enteral diets on the injury and inflammation observed in the colon, liver and spleen.

DEPR:
To assess the effects of SAZ in this model, female Lewis rats were orally administered SAZ immediately following the induction of colitis. Rats were given chow ad libitum for the duration of the four week study period. Similar measurements were assessed as described below.

DEPR:
Recent studies have suggested that nutritional supplementation in the form of enteral diets may prove useful as adjunctive or primary therapy for patients with IBD. Indeed, recent reports suggest that n-3 fatty acids from fish oil as well as the SCFA produced during the fermentation of indigestible carbohydrates may attenuate some of the pathophysiology associated with active gut inflammation. Therefore, we ascertained whether three enteral diets, one supplemented with fish oil or two different diets supplemented with two forms of indigestible carbohydrate could inhibit some of the inflammation observed in a model of chronic colitis. The results are presented in FIGS. 1-5. FIG. 1 presents colon weights of animals following the various therapies (diets). FIG. 2 presents MPO activity in colonic tissue of rats following the various therapies (diets). FIG. 3 presents liver weights of animals following the various therapies (diets). FIG. 4 presents spleen weights in the animals following the various therapies (diets). FIG. 5 presents levels in circulating plasma of nitrate and nitrite in animals following the various therapies (diets).

DETL:
TABLE 5 SHORT CHAIN FATTY ACID PRODUCTION DURING 3, 6, 12 AND 24 H in vitro FERMENTATION OF VARIOUS OLIGOSACCHARIDES SUB- SHORT CHAIN FATTY ACID.sup.a Total STRATE HOUR Acetate Propionate Butyrate Lactate SCFA.sup.b FOS 3 1.49 .20 .23 .45 2.37 6 3.61 .54 .87 1.19 6.21 12 3.67 1.01 1.64 .54 6.86 24 3.20 1.09 2.09 .01 6.39 Rafti- 3 1.42 .20 .27 .47 2.36 lose .RTM. 6 3.49 .53 .92 1.28 6.22 12 3.68 .98 1.70 .59 6.95 24 3.09 1.05 2.1 .01 6.30 XOS 3 1.21 .15 .13 .14 1.63 6 4.12 .58 .58 .47 5.75 12 5.90 .97 1.1 .74 8.72 24 5.53 .96 1.5 .05 8.10 Statistics SEM .13 .08 .08 .08 LSD.sup.c .37 .23 .23 .23

.sup.a Calculated as (mmol fatty acid in incubation tube minus mmol fatty acid in blank tube) divided by original

substrate dry matter (DM) and expressed as mmol/g substrate DM. .sup.b Sum of acetate + propionate + butyrate + lactate and expressed as mmol/g substrate DM. .sup.c Differences between mean values within a column greater than the specified LSD are significantiy different P < .05.

ORPL:
Scheppach, et al., "Effect of Butyrate Enemas on the Colonic Mucosa in Distal Ulcerative Colitis," Gastroenterology, 103:51-56, 1992.

ORPL:
Vilaseca, et al., "Dietary Fish Oil Reduces Progression of Chronic Inflammatory Lesions in a Rat Model of Granulomatous Colitis," Gut, 31:539, 1990.

☐ 5. Document ID: US 5780451 A

L33: Entry 5 of 8 File: USPT Jul 14, 1998

US-PAT-NO: 5780451
DOCUMENT-IDENTIFIER: US 5780451 A

TITLE: Nutritional product for a person having ulcerative colitis

DATE-ISSUED: July 14, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
DeMichele; Stephen Joseph	Dublin	OH		
Garleb; Keith Allen	Powell	OH		
McEwen; John William	Gahanna	OH		
Fuller; Martha Kay	Westerville	OH		

US-CL-CURRENT: 514/54; 426/567, 426/658, 514/168, 514/188, 514/552, 514/566, 514/725, 514/810, 514/812, 514/813, 514/861

ABSTRACT:

An enteral nutritional product for a person having ulcerative colitis contains in combination (a) an oil blend which contains eicosapentaenoic acid (20:5n3) and/or docosahexaenoic acid (22:6n3), and (b) a source of indigestible carbohydrate which is metabolized to short chain fatty acids by microorganisms present in the human colon. Preferably the nutritional product also contains one or more nutrients which act as antioxidants.

18 Claims, 5 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 5

L33: Entry 5 of 8 File: USPT Jul 14, 1998

DOCUMENT-IDENTIFIER: US 5780451 A
TITLE: Nutritional product for a person having ulcerative colitis

BSPR:
Increasing interest has been generated in the use of enemas/irrigation solutions containing buffered, physiologic levels of SCFAs for the treatment of diversion colitis and ulcerative colitis. Diversion colitis is an inflammatory process arising in segments of the colorectum at various intervals after surgical diversion of the fecal stream. The endoscopic appearance is similar to those of

active Crohn's Disease and ulcerative colitis. Glotzer et al., "Proctitis and Colitis Following Diversion of the Fecal Stream", GASTROENTEROLOGY Vol. 80, pages 438-441 (1981). The cause of this condition is not known, but one mechanism has been postulated; a nutritional deficiency of the colonic epithelium, specifically due to the absence of SCFAs normally present in colonic contents, Komorowski, "Histologic Spectrum of Diversion Colitis" AMERICAN JOURNAL OF SURGICAL PATHOLOGY, Vol. 14, page 548 (1990), Roediger, "The Starved Colon--Diminished Mucosal Nutrition, Diminished Absorption, and Colitis", DISEASES OF THE COLON AND RECTUM, Vol. 33, pages 858-862 (1990). Harig et al., "Treatment of Diversion Colitis with Short-Chain-Fatty Acid Irrigation", NEW ENGLAND JOURNAL OF MEDICINE, Vol. 320, No. 1, pages 23-28 (1989) tested this hypothesis by assessing whether irrigation with SCFAs could ameliorate inflammation in four patients with diversion colitis. These patients were administered SCFAs twice daily for 2-3 weeks with 60 mL of an enema solution comprising a physiologic mixture of SCFAs as sodium salts. After 2-3 weeks of therapy, macroscopic and histological resolution of inflammation was evident. An impaired utilization of SCFAs has also been implicated in ulcerative colitis which suggests that diminished intracellular energy production may be important in the inflammatory process, Roediger, "The Colonic Epithelium in Ulcerative Colitis: an Energy Deficiency Disease", THE LANCET, Oct. 4, 1980, pages 712-715. Vernia et al., "Fecal Lactate and Ulcerative Colitis", GASTROENTEROLOGY, Vol. 95, pages 1564-1568 (1988); and Vernia et al., "Organic Anions and the Diarrhea of Inflammatory Bowel Disease", DIGESTIVE DISEASES AND SCIENCES, Vol. 33, pages 1353-1358 (1988) have shown that fecal water from patients with ulcerative colitis contains reduced concentrations of SCFAs as well as markedly increased lactate and low pH. In a study by Breuer et al., "Rectal Irrigation with Short-Chain Fatty Acids for Distal Ulcerative Colitis" (preliminary report), DIGESTIVE DISEASES AND SCIENCES, Vol. 36, pages 185-187 (1991), relates an investigation of large bowel irrigation with SCFAs in patients with ulcerative colitis. It was found that 9 out of 10 patients completing the study were judged to be at least much improved and showed a significant change in mean disease activity index score and mucosal histology score. Recently Senagore et al., "Short-Chain Fatty Acid Enemas: a Cost Effective Alternative in the Treatment of Nonspecific Proctosigmoiditis", DISEASES OF THE COLON AND RECTUM, Vol. 35, page 923 (1992), confirmed the results of Breuer et al. demonstrating an 80 percent response rate in patients with idiopathic proctosigmoiditis. This study indicates that administering a solution of SCFAs similar to Harig et al. for six weeks was equally efficacious to corticosteroid or 5-ASA enemas for the treatment of proctosigmoiditis at a significant cost savings. Scheppach et al., "Effect of Butyrate Enemas on the Colonic Mucosa in Distal Ulcerative Colitis", GASTROENTEROLOGY, Vol. 103, pages 51-56 (1992) investigated the use of butyrate enemas alone rather than the SCFA mixture to treat ten patients with distal ulcerative colitis in a placebo-controlled, single-blind, randomized trial. The authors concluded that markedly improved disease activity index and histological parameters suggesting that the effect of a SCFA mixture on the inflamed mucosa in ulcerative colitis is largely attributable to its butyrate moiety.

BSPR:
Certain of the organisms that inhabit the large bowel can utilize dietary fiber (eg, pectin and gum arabic) and indigestible oligosaccharides (eg, fructooligosaccharides and xylooligosaccharides) as an energy source. Smith et al., "Introduction to Metabolic Activities of Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 32, pages 149-157 (1979); Miller et al., "Fermentation by Saccharolytic Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 32, pages 164-172 (1979); Cummings., "Fermentation in the Human Large Intestine: Evidence and Implications for Health", THE LANCET, May 28, 1983 pages 1206-1209 Titgemeyer et al., "Fermentability of Various Fiber Sources by Human Fecal Bacteria In Vitro", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 53, pages 1418-1424 (1991). The microorganisms derive energy from the carbohydrate sources through a process referred to as anaerobic fermentation. During fermentation, the microorganisms produce SCFAs (eg, acetate, propionate, butyrate) as the major end products. Salyers et al., "Fermentation of Mucin and Plant Polysaccharides by Strains of Bacteroides from the Human Colon", APPLIED AND ENVIRONMENTAL MICROBIOLOGY, Vol. 33, No. 2, pages 319-322 (1977); Mitsuoka et al., "Effect of Fructo-oligosaccharides on

Intestinal Microflora", DIE NAHRUNG, Vol. 31, pages 427-436 (1987); Tokunaga et al., "Influence of Chronic Intake of New Sweetener Fructooligosaccharide (Neosugar) on growth and Gastrointestinal Function of the Rat", JOURNAL OF NUTRITIONAL SCIENCE AND VITAMINOLOGY, Vol. 32, pages 111-121 (1986).

DEPR:
Analysis of acetate, propionate and butyrate was conducted according to Merchen et al., "Effect of Intake and Forage Level on Ruminal and Turnover Rates, Bacterial Protein Synthesis and Duodenal Amino Acid Flows in Sheep", JOURNAL OF ANIMAL SCIENCE, Vol. 62, pages 216-225 (1986). Briefly, an aliquot from the balch tube was acidified with 6N HCl and centrifuged at 31,000.times.g for 20 minutes Concentrations of acetate, propionate and butyrate were determined in the supernatant using a Hewlett-Packard 5890A gas chromatograph and a column (180 cm.times.4 mm id) packed with 20% Tween 80-2% H.sub.3 PO.sub.4 on 60 to 80 mesh Chromosorb W (Supelco Inc, Bellefonte, Pa., U.S.A.). Nitrogen was used as a carrier gas with a flow rate of 70 mL/minutes. Oven temperature was 120.degree. C. and detector and injector temperatures were 200.degree. C. Lactate was determined calorimetrically using a method described in Barker et al., "The Colorimetric Determination of Lactic Acid in Biological Material", JOURNAL OF BIOLOGICAL CHEMISTRY, Vol. 138, page 535, (1941).

DEPR:
SCFA production (acetate, propionate, butyrate and lactate) during in vitro fermentation of the oligosaccharides is presented in Table 3. Four time points were studied and include 3, 6, 12 and 24 hours. Retention time in the large bowel of humans will dictate the length of fermentation in vivo. In cases where retention time is great, the extent of substrate fermentability will be a factor which most influences SCFA production. If retention time is short, the rate of substrate fermentation becomes more important. Since retention times can differ significantly in an in vivo situation it is necessary to monitor substrate degradation over time in vitro in order that comparisons can be made.

DEPR:
Fermentation of all oligosaccharides was rapid, essentially being complete by 6 hours for the fructooligosaccharides (FOS and Raftilose) and by 12 hours for XOS. The results are presented in Table 5. It is recommended that the 6 hour and 12 hour values be used to estimate the composition of the end-products for the fructooligosaccharides and the XOS, respectively, even though retention times in the large bowel can be considerably longer. At later time points it becomes apparent that lactate is being converted to propionate and acetate to butyrate. Interconversion in a closed in vitro system can be a problem with rapidly fermented substrates. It does not reflect the true state of the large bowel where the fatty acids are continually absorbed.

DEPR:
As is typically found with in vitro fermentations using human fecal inoculum or in analysis of fecal samples, acetate was the short chain fatty acid found in the highest concentration. Titgemeyer et al., "Fermentability of Various Fiber Sources by Human Fecal Bacteria In Vitro", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 53, Pages 1418-1424, (1991). Baldwin., "Energy Metabolism in Anaerobes", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 23, No. 11, pages 1508-1513, (1970) determined that acetate, propionate and butyrate account for 83% of the SCFAs produced during anaerobic fermentation by large bowel microflora, and the remaining SCFAs are distributed among isovaleric, isobutyric, valeric, lactic, formic and succinic acids. In this study, a considerable amount of lactate was found, particularly during fermentation with FOS and Raftilose. It has been documented that the oligosaccharides used in this study serve as an energy source for Bifidobacteria and that there consumption will lead to the selective growth of this organism in the GI tract. Okazaki et al., "Effects of Xylooligosaccharides on the Growth of Bifidobacteria", BIFIDOBACTERIA MICROFLORA, Vol. 9, No. 2 page 77, (1990); Mitsuoka et al., "Effects of Fructo-oligosaccharide on Intestinal Microflora", DIE NAHRUNG, Vol. 31, pages 427-436, (1987). The primary end products produced by Bifidobacteria during fermentation are acetate and lactate. Miller et al., "Fermentations by Saccharolytic Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol.

32, pages 164-172, (1979). The fact that these oligosaccharides serve as an energy source for the Bifidobacteria could explain the elevated levels of lactate found in this study.

DEPR:
Total short-chain fatty acid production was greater for the xylooligosaccharides (XOS) compared to the fructooligosaccharides (FOS and Raftilose). The primary factor effecting the quantity of SCFAs produced during fermentation is the fermentability of the substrate. It is assumed that the oligosaccharides are completely fermented in this system. However, the yield of SCFAs (mol) from a substrate is dependent not only on the weight of the substrate fermented but also on the average molecular weight of the oligosaccharide component sugars. One can assume that the fermentation of one monosaccharide molecule can result in either two acetate, two propionate, two lactate or one molecule of butyrate. The molecular weight of the components of the fructooligosaccharides (glucose and fructose, 180) is greater than the molecular weight of xylose (150) which is the monomeric component of XOS. Subsequently, on an equivalent weight basis, there are more moles of monosaccharide molecules with the xylooligosaccharide compared to the fructooligosaccharide. This would explain the greater production of SCFA with the XOS compared to the fructooligosaccharides. Lastly, the quantity and profile of SCFAs produced was virtually identical between the two fructooligosaccharides (Raftilose and FOS). While these fructooligosaccharides differ to some extent in their chemical composition, it is apparent that they are metabolized similarly in this in vitro fermentation system.

DEPR:
Recent evidence that the regular intake of n-3 fatty acids from fish oil inhibits neutrophil and monocyte functions suggests that n-3 fatty acids have antiinflammatory properties. Beneficial effects of marine lipids have been shown in animal models of inflammatory bowel disease. Empey et al., "Fish Oil-Enriched Diet is Mucosal Protective Against Acetic Acid-Induced Colitis in Rats", CANADIAN JOURNAL OF PHYSIOLOGY AND PHARMACOLOGY, Vol. 69, pages 480-487 (1991); Vilaseca et al., "Dietary Fish Oil Reduces Progression of Chronic Inflammatory Lesions in a Rat Model of Granulomatous Colitis", , GUT, Vol. 31, page 539 (1990). In preliminary therapeutic trials, diet supplementation with fish oil has led to symptomatic improvement of patients with ulcerative colitis, and reduced ethanol-induced damage in human duodenal mucosa. Schepp et al., "Fish Oil Reduces Ethanol-Induced Damage of the Duodenal Mucosa in Humans", EUROPEAN JOURNAL OF CLINICAL INVESTIGATION, Vol. 21, pages 230-237 (1991) Lorenz et al., "Supplementation with n-3 Fatty Acids from Fish Oil in Chronic Inflammatory Bowel Disease", JOURNAL OF INTERNAL MEDICINE SUPPLEMENT, Vol. 225, pages 225-232 (1989); Hillier et al., "Incorporation of Fatty Acids from Fish Oil and Olive Oil into Colonic Mucosal Lipids and Effects Upon Eicosanoid Synthesis in Inflammatory Bowel Disease", GUT, Vol. 32, pages 1151-1155 (1991); Saloman et al., "Treatment of Ulcerative Colitis with Fish Oil N-3-w-Fatty Acid: An Open Trial", JOURNAL OF CLINICAL GASTROENTEROLOGY, Vol. 12, No. 2, pages 157-161 (1990).

DEPR:
A major limitation in investigating the pathogenic mechanisms responsible for the mucosal injury observed during chronic inflammation of the intestine and colon has been the relative paucity of relevant animal models. Two models of colitis produced in rats that have received much attention over the past few years are the acetic acid and trinitrobenzene sulfonic acid (TNBS) models. The mechanism by which acetic acid produces the diffuse colitis is thought to involve nonspecific, acid induced injury to the colonic mucosa that is followed by an acute inflammatory response. Apparently the protonated form of the acid is required to induce the colitis since neither HCl (pH 2.3) nor sodium acetate (pH 7.0) is effective in eliciting the inflammatory response. However, there is some evidence to suggest that acetic acid may promote other pathophysiological events (e.g. fluid and electrolyte secretion) using noncytotoxic concentrations of the acid.

DEPR:
Recent studies have demonstrated that the intrarectal administration of the

hapten, TNBS, in the presence of a mucosal barrier breaker such as ethanol, produces an acute and possibly chronic colitis in unsensitized rats. The mechanism(s) by which buffered or unbuffered TNBS in the presence of ethanol initiates inflammation in unsensitized animals is unclear; however, it has been suggested to involve macrophage-mediated recognition and lysis of TNBS-modified autologous cells within the mucosa. However, more recent evidence suggests more complicated mechanisms. For example, the barrier breaker, ethanol, is an extremely potent pro-inflammatory solvent alone. Furthermore, it has been demonstrated that TNBS is metabolized by certain colonic enzymes and substrates to yield both pro-inflammatory and cytotoxic oxidants that could initiate colonic inflammation. Grisham et al., "Metabolism of Trinitrobenzene Sulfonic Acid by the Rat Colon Produces Reactive Oxygen Species", GASTROENTEROLOGY, Vol. 101, pages 540-547 (1991). A recent study directly compared the acetic acid and the TNBS (+ETOH) models of colitis and found that either model may be useful to study those events that occur at the time of inflammation (e.g. arachidonate metabolism, granulocyte infiltration and metabolism, etc.) or during repair. However, the use of these models of colitis may have significant limitations in understanding those immunological events that initiate the acute and chronic inflammatory episodes. For example, the inflammation and tissue injury observed in human inflammatory bowel disease is most probably a result of inappropriate immunological activation (e.g. autoimmune, infectious agent, etc.) whereas the inflammation induced by the intrarectal application of acetic acid, ethanol or ethanol plus TNBS is a response to extensive mucosal injury. Thus, the mechanisms by which inflammation (and mucosal injury) are achieved in the human disease may be very different than those in the experimental models.

DEPR:
For these reasons, a model of acute and chronic distal colitis in rats was developed based upon a previously published method in which purified bacterial cell wall polymers (derived from Group A streptococci) are injected intramurally into the distal colon of genetically-susceptible rats. Sartor et al., "Granulomatous Entercolitis Induced by Purified Bacterial Cell Wall Fragments", GASTROENTEROLOGY, Vol. 89, pages 587-595 (1985). This model produces an acute and chronic inflammation characterized by the infiltration of large numbers of inflammatory cells, enhanced mucosal permeability, interstitial fibrosis, and mucosal thickening as well as the extraintestinal manifestations of arthritis, hepatic and splenic granulomas. Unlike most models of colitis, the inflammation induced in this model promoting mucosal and submucosal injury rather than the injury causing the inflammation.

DEPR:
In Experiment 2 the objectives were: (a) to determine whether this model of colitis responds to sulfasalazine (SAZ) and (b) to assess the effects of specially formulated enteral diets on the injury and inflammation observed in the colon, liver and spleen.

DEPR:
To assess the effects of SAZ in this model, female Lewis rats were orally administered SAZ immediately following the induction of colitis. Rats were given chow ad libitum for the duration of the four week study period. Similar measurements were assessed as described below.

DEPR:
Recent studies have suggested that nutritional supplementation in the form of enteral diets may prove useful as adjunctive or primary therapy for patients with IBD. Indeed, recent reports suggest that n-3 fatty acids from fish oil as well as the SCFA produced during the fermentation of indigestible carbohydrates may attenuate some of the pathophysiology associated with active gut inflammation. Therefore, we ascertained whether three enteral diets, one supplemented with fish oil or two different diets supplemented with two forms of indigestible carbohydrate could inhibit some of the inflammation observed in a model of chronic colitis. The results are presented in FIGS. 1-5. FIG. 1 presents colon weights of animals following the various therapies (diets). FIG. 2 presents MPO activity in colonic tissue of rats following the various therapies (diets). FIG. 3 presents liver weights of animals following the

various therapies (diets). FIG. 4 presents spleen weights in the animals following the various therapies (diets). FIG. 5 presents levels in circulating plasma of nitrate and nitrite in animals following the various therapies (diets).

DETL:
TABLE 5 _____ SHORT CHAIN FATTY ACID PRODUCTION DURING 3, 6, 12 AND 24 H in vitro FERMENTATION OF VARIOUS OLIGOSACCHARIDES SHORT CHAIN FATTY ACID.sup.a SUB- Propi- Total STRATE HOUR Acetate onate Butyrate Lactate SCFA.sup.b _____ FOS 3 1.49 .20 .23 .45 2.37 61 3.61 .54 .87 1.19 6.21 12 3.67 1.01 1.64 .54 6.86 24 3.20 1.09 2.09 .01 6.39 Raftilose .RTM. 3 1.42 .20 .27 .47 2.36 6 3.49 .53 .92 1.28 6.22 12 3.68 .98 1.70 .59 6.95 24 3.09 1.05 2.1 .01 6.30 XOS 3 1.21 .15 .13 .14 1.63 6 4.12 .58 .58 .47 5.75 12 5.90 .97 1.1 .74 8.72 24 5.53 .96 1.5 .05 8.10 Statistics SEM .13 .08 .08 .08 LSD.sup.c .37 .23 .23 .23
_____ .sup.a Calculated as (mmol fatty acid in incubation tube minus mmol fatty acid in blank tube) divided by original substrate dry matter (DM) and expressed as mmol/g substrate DM. .sup.b Sum of acetate + propionate + butyrate + lactate and expressed as mmol/g substrate DM. .sup.c Differences between mean values within a column greater than the specified LSD are significantly different P < .05.

ORPL:
Scheppach et al., "Effect of Butyrate Enemas on the Colonic Mucosa in Distal Ulcerative Colitis", Gastroenterology, 103:51-56 (1992).

ORPL:
Vilaseca et al., "Dietary Fish Oil Reduces Progression of Chronic Inflammatory Lesions in a Rat Model of Granulomatous Colitis", Gut, 31:539 (1990).

☐ 6. Document ID: US 5733579 A

L33: Entry 6 of 8 File: USPT Mar 31, 1998

US-PAT-NO: 5733579
DOCUMENT-IDENTIFIER: US 5733579 A

TITLE: Oral rehydration solution containing indigestible oligosaccharides

DATE-ISSUED: March 31, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Wolf; Bryan Warren	Newark	OH		
Garleb; Keith Allen	Powell	OH		
Campbell; Sheila Martinson	Worthington	OH		
Meulbroek; Jonathan Allan	Grayslake	IL		
Wheeler; Keith Brian	Dublin	OH		
Walton; Joseph Edward	Westerville	OH		

US-CL-CURRENT: 424/606; 424/610, 424/663, 424/717, 514/23, 514/867

ABSTRACT:

An oral rehydration solution contains indigestible oligosaccharides. Diarrhea related dehydration requires fluid and electrolyte replacement. The primary etiology of antibiotic-associated diarrhea (also known as pseudomembranous colitis) has been recognized as Clostridium difficile. It is believed that the indigenous microflora of a healthy individual suppresses the normally present C. difficile. However, when the indigenous microflora are disrupted (e.g., during antibiotic treatment) overgrowth of C. difficile may occur causing diarrhea and colitis. Treatment of diarrhea related to C. difficile with rehydration therapy and antibiotics has proven effective, but many times relapse occurs. It has been suggested that normalization of the microflora will inhibit C. difficile relapse. Indigestible oligosaccharides have been shown to inhibit C. difficile infection.

8 Claims, 12 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 8

L33: Entry 6 of 8 File: USPT Mar 31, 1998

DOCUMENT-IDENTIFIER: US 5733579 A
TITLE: Oral rehydration solution containing indigestible oligosaccharides

DEPR:
It is becoming increasingly obvious that many of the beneficial effects of fermentable carbohydrate are mediated by short chain fatty acids (SCFA) such as acetate, propionate, and butyrate, which are produced during anaerobic fermentation in the colon. Short chain fatty acids play a key role in bowel function. The absorption of 100 mmole SCFA is associated with the absorption of 360 ml water. Casparey et al., "Bacterial fermentation of carbohydrates within the gastrointestinal tract", CLINICAL RESEARCH REVIEW, (Suppl. 1):107-177 (1981). Subsequently, the absence or reduction of SCFA in the colon could result in diarrhea. Ramakrishna et al. "Colonic dysfunction in acute diarrhea: the role of luminal short chain fatty acids", GUT, 34:1215 (1993) found that fecal output of short chain fatty acids in patients with acute diarrhea was low on the first day of illness, but increased over the next five days as the patients condition improved. Further, using an in vivo rectal dialysis technique, Ramakrishna et al. "Colonic dysfunction in acute diarrhoea: the role of luminal short chain fatty acids", GUT, 34:1215 (1993) demonstrated that lumenal SCFA could restore net water and sodium reabsorption in the rectum of patients with acute diarrhea. In vivo perfusion studies in healthy subjects have shown secretion of salt and water in the ascending colon in response to enteral feeding. Bowling et al., "Colonic secretory effect in response to enteral feeding in man", GUT, 34(suppl. 1):A54 (1993); Bowling et al., "The colonic secretory response to enteral feeding:influence of high strength diet", CLINICAL NUTRITION, 12(suppl. 2):23

(1993). Bowling et al., "Reversal by short-chain fatty acids of colonic fluid secretion induced by enteral feeding", THE LANCET, 342:1266 (1993) investigated the effect of short chain fatty acids on colonic fluid secretion induced by enteral feed. The researchers found that SCFA infusion directly into the cecum of healthy subjects reversed the fluid secretion seen in the ascending colon during enteral feeding and theorized that these findings could have implications for the management of diarrhea related to enteral feedings.

DEPR:
Two experiments were conducted to determine the effect of fructooligosaccharides (FOS) as a dietary supplement on mortality in a Syrian hamster model of C. difficile-colitis.

DEPR:
The use of the Syrian hamster as a model for C. difficile-colitis is widely recognized. Lust et al., "Clindamycin-Induced Enterocolitis in Hamsters", THE JOURNAL OF INFECTIOUS DISEASES, 137(4) 464-475 (1978), proposed that the enterocolitis induced in the hamster by antibiotics is a good model for investigation of the syndrome in humans. Price et al., "Morphology of experimental antibiotic-associated enterocolitis in the hamster: a model for human pseudomembranous colitis and antibiotic-associated diarrhoea", GUT, 20:467-475 (1979) set out to study the morphology of experimental antibiotic-associated pseudomembranous colitis (PMC). They noted that the hamster model has some morphological differences; however, the bacteriology and toxicology are identical to the human. They concluded that the hamster is a good model for investigating the pathogenesis of PMC and antibiotic-associated enteropathy in general. In fact, Wilson et at., "Suppression of Clostridium difficile by Normal Hamster Cecal Flora and Prevention of Antibiotic-Associated Cecitis", INFECTION AND IMMUNITY, 34(2) 626-628 (1981), noted that studies with the hamster model of antibiotic-associated colitis led to the discovery of C. difficile toxin as a major etiology of antibiotic-associated colitis in humans and to effective treatment with oral vancomycin. Wilson et al., "Population Dynamics of Ingested Clostridium difficile in the Gastrointestinal Tract of the Syrian Hamster", THE JOURNAL OF INFECTIOUS DISEASES, 151(2)355-361 (1985) noted that the best studied animal model of antibiotic-associated colitis was that of the Syrian hamster.

DEPR:
Median survival time for treatment groups are presented in Table 3. As in the previous experiment, the overall effect of FOS was to increase MST of hamsters (P less than 0.05). In this experiment, there appeared to be an interaction between the Vancomycin treatment and inoculation level (Table 4). Hamsters supplemented with FOS and inoculated with C. difficile but not treated with Vancomycin tended to have improved MST (P less than 0.10). However, FOS had no effect (P greater than 0.20) on inoculated hamsters treated with Vancomycin. On the other hand, FOS supplementation increased MST of non-inoculated hamsters treated or not treated with Vancomycin (P less than 0.05 and P less than 0.01, respectively; survival curves, FIGS. 5 and 6, respectively). Again, survival curves show the added benefits of FOS. Overall, hamsters fed FOS had increased (P less than 0.05) MST. These data suggest that dietary supplementation with FOS increases median survival time in a hamster model of Clostridium difficile-colitis.

DEPR:
In general, the results of Experiments 1 and 2 show that enteral administration of a therapeutically effective amount of an indigestible oligosaccharide (FOS) inhibits the infection of a mammal by C. difficile as shown by improved survival times in a hamster model of C. difficile-colitis. This has been shown by an increase in survivability (in certain treatment groups) and a consistent increase in median survival time. Impressively, the improved survival time due to FOS supplementation was above the effect of fiber contained in the chow diet.

☐ 7. Document ID: US 5712274 A

L33: Entry 7 of 8 File: USPT Jan 27, 1998

US-PAT-NO: 5712274
DOCUMENT-IDENTIFIER: US 5712274 A

TITLE: Thienotriazolodiazepine compounds and their pharmaceutical use

DATE-ISSUED: January 27, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Sueoka; Hiroyuki	Fukuoka			JPX
Ehara; Shuji	Fukuoka			JPX
Kobayashi; Haruhito	Fukuoka			JPX
Arichi; Takeshi	Fukuoka			JPX
Komatsu; Hirotsugu	Saitama			JPX

US-CL-CURRENT: 514/219; 514/220, 540/555, 540/560

ABSTRACT:

Thienotriazolodiazepine compounds of the formula (1) ##STR1## wherein each symbol is as defined in the specification, pharmaceutically acceptable salts thereof, and pharmaceutical use thereof. The compounds of the present invention are useful as preventive and therapeutic drugs for inflammatory diseases and allergic diseases, in which cell adhesion is involved.

12 Claims, 0 Drawing figures Exemplary Claim Number: 1

L33: Entry 7 of 8 File: USPT Jan 27, 1998

DOCUMENT-IDENTIFIER: US 5712274 A
TITLE: Thienotriazolodiazepine compounds and their pharmaceutical use

BSPR:
Effect on trinitrobenzenesufonic acid (TNBS)-induced colitis model in rats

BSPR:
Effect on trinitrobenzenesufonec acid (TNBS)-induced colitis model in rats.

DEPR:
4-(4-Chlorophenyl)-2-ethyl-9-methyl-6H-thieno[3,2-f][1,2,4]tria zolo[4,3-a][1,4]diazepine (6 g) is dissolved in diethyl carbonate (100 ml) under a nitrogen stream, and 60% sodium hydride (1.2 g) is added with stirring at room temperature. The mixture is refluxed under heating for 1 hour and cooled to 50.degree. C. with ice water. Ethyl bromobutyrate (4.3 ml) is added. After stirring at 100.degree. C. for 2 hours, the reaction mixture is poured into ice water (1 l) and extracted with ethyl acetate. The organic layer is washed with saturated brine, dried over anhydrous magnesium sulfate and concentrated under reduced pressure. The residue is purified by silica gel column chromatography (ethyl acetate:methanol=100:1) and crystallized from isopropyl ether to give 3.9 g of (.+-.)-ethyl
4-(4-(4-chlorophenyl)-6-ethoxycarbonyl-2-ethyl-9-methyl-6H-thieno[3,2-f][1,2,4]triazolo[4,3-a][1,4]diazepin-6-yl)butyrate as white powdery crystals.
(.+-.)-Ethyl
4-(4-(4-chlorophenyl)-6-ethoxycarbonyl-2-ethyl-9-methyl-6H-thieno[3,2-f][1,2,4]-triazolo[4,3-a][1,4]diazepin-6-yl)butyrate (3.5 g) is dissolved in a mixture of ethanol (180 ml) and water (60 ml), and barium hydroxide 8 hydrate (8.4 g) is added. The mixture is refluxed under heating for 4 hours. After the completion of the reaction, ethanol is distilled away under reduced pressure and adjusted to pH 1 with 1N hydrochloric acid. The mixture is stirred for 30 minutes and adjusted to pH 4 with a saturated aqueous solution of sodium hydrogencarbonate to give 1.1 g of
(.+-.)-4-(4-(4-chlorophenyl)-2-ethyl-9-methyl-6H-thieno[3,2-f][1,2,4]triazolo[4,3-a][1,4]diazepin-6-yl)butyric acid as white powdery crystals, melting point 185.degree.-187.degree. C.

☐ 8. Document ID: US 5444054 A

L33: Entry 8 of 8 File: USPT Aug 22, 1995

US-PAT-NO: 5444054
DOCUMENT-IDENTIFIER: US 5444054 A

TITLE: Method of treating ulcerative colitis

DATE-ISSUED: August 22, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Garleb; Keith A.	Powell	OH		
Demichele; Stephen J.	Dublin	OH		

US-CL-CURRENT: 514/54; 426/72, 514/867, 514/925

ABSTRACT:

A method of improving the nutritional status and reversing the characteristic diarrhea and inflammatory condition in a mammalian creature having ulcerative colitis or inflammation of the colon which contains in combination (a) an oil blend which contains eicosapentaenoic acid (20:5n3) and/or docosahexaenoic acid (22:6n3), and (b) a source of indigestible carbohydrate which is metabolized to short chain fatty acids by microorganisms present in the human colon. Preferably the nutritional product also contains one or more nutrients which act as antioxidants.

19 Claims, 5 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 5

L33: Entry 8 of 8 File: USPT Aug 22, 1995

DOCUMENT-IDENTIFIER: US 5444054 A
TITLE: Method of treating ulcerative colitis

BSPR:
Increasing interest has been generated in the use of enemas/irrigation solutions containing buffered, physiologic levels of SCFAs for the treatment of diversion colitis and ulcerative colitis. Diversion colitis is an inflammatory process arising in segments of the colorectum at various intervals after surgical diversion of the fecal stream. The endoscopic appearance is similar to those of active Crohn's Disease and ulcerative colitis. Glotzer et al., "Proctitis and Colitis Following Diversion of the Fecal Stream", GASTROENTEROLOGY Vol. 80, pages 438-441 (1981). The cause of this condition is not known, but one mechanism has been postulated; a nutritional deficiency of the colonic epithelium, specifically due to the absence of SCFAs normally present in colonic contents, Komorowski, "Histologic Spectrum of Diversion Colitis" AMERICAN JOURNAL OF SURGICAL PATHOLOGY, Vol. 14, page 548 (1990), Roediger, "The Starved Colon--Diminished Mucosal Nutrition, Diminished Absorption, and Colitis", DISEASES OF THE COLON AND RECTUM, Vol. 33, pages 858-862 (1990). Harig et al., "Treatment of Diversion Colitis with Short-Chain-Fatty Acid Irrigation", NEW ENGLAND JOURNAL OF MEDICINE, Vol. 320 No. 1, pages 23-28 (1989) tested this hypothesis by assessing whether irrigation with SCFAs could ameliorate inflammation in four patients with diversion colitis. These patients were administered SCFAs twice daily for 2-3 weeks with 60 mL of an enema solution comprising a physiologic mixture of SCFAs as sodium salts. After 2-3 weeks of therapy, macroscopic and histological resolution of inflammation was evident. An impaired utilization of SCFAs has also been implicated in ulcerative colitis which suggests that diminished intracellular energy production may be important in the inflammatory process, Roediger, "The Colonic Epithelium in Ulcerative Colitis: an Energy Deficiency Disease?", THE LANCET, Oct. 4, 1980, pages 712-715 (1980). Vernia et al., "Fecal Lactate and Ulcerative Colitis", GASTROENTEROLOGY, Vol. 95, pages 1564-1568; and Vernia et al., "Organic Anions and the Diarrhea of Inflammatory Bowel Disease", DIGESTIVE DISEASES AND SCIENCES, Vol. 33, pages 1353-1358 (1988) have shown that fecal water from patients with ulcerative

colitis contains reduced concentrations of SCFAs as well as markedly increased lactate and low pH. In a study by Breuer et al., "Rectal Irrigation with Short-Chain Fatty Acids for Distal Ulcerative Colitis" (preliminary report), DIGESTIVE DISEASES AND SCIENCES, Vol. 36, pages 185-187 (1991), relates an investigation of large bowel irrigation with SCFAs in patients with ulcerative colitis. It was found that 9 out of 10 patients completing the study were judged to be at least much improved and showed a significant change in mean disease activity index score and mucosal histology score. Recently Senagore et al., "Short-Chain Fatty Acid Enemas: a Cost Effective Alternative in the Treatment of Nonspecific Proctosigmoiditis", DISEASES OF THE COLON AND RECTUM, Vol. 35, page 923 (1992), confirmed the results of Breuer et al. demonstrating an 80 percent response rate in patients with idiopathic proctosigmoiditis. This study indicates that administering a solution of SCFAs similar to Harig et al. for six weeks was equally efficacious to corticosteroid or 5-ASA enemas for the treatment of proctosigmoiditis at a significant cost savings. Scheppach et al., "Effect of Butyrate Enemas on the Colonic Mucosa in Distal Ulcerative Colitis", GASTROENTEROLOGY, Vol. 103, pages 51-56 (1992) investigated the use of butyrate enemas alone rather than the SCFA mixture to treat ten patients with distal ulcerative colitis in a placebo-controlled, single-blind, randomized trial. The authors concluded that markedly improved disease activity index and histological parameters suggesting that the effect of a SCFA mixture on the inflamed mucosa in ulcerative colitis is largely attributable to its butyrate moiety.

BSPR:
Certain of the organisms that inhabit the large bowel can utilize dietary fiber (eg, pectin and gum arabic) and indigestible oligosaccharides (eg, fructooligosaccharides and xylooligosaccharides) as an energy source. Smith et al., "Introduction to Metabolic Activities of Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 32, pages 149-157 (1979); Miller et al., "Fermentation by Saccharolytic Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 32, pages 164-172 (1979); Cummings, "Fermentation in the Human Large Intestine: Evidence and Implications for Health", THE LANCET, May 28, 1983, pages 1206-1209; Titgemeyer et al., "Fermentability of Various Fiber Sources by Human Fecal Bacteria In Vitro", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 53, pages 1418-1424 (1991). The microorganisms derive energy from the carbohydrate sources through a process referred to as anaerobic fermentation. During fermentation, the microorganisms produce SCFAs (eg, acetate, propionate, butyrate) as the major endproducts. Salyers et al., "Fermentation of Mucin and Plant Polysaccharides by Strains of Bacteroides from the Human Colon", APPLIED AND ENVIRONMENTAL MICROBIOLOGY, Vol. 33, No. 2, pages 319-322 (1977); Mitsuoka et al., "Effect of Fructo-oligosaccharides on Intestinal Microflora", DIE NAHRUNG, Vol. 31, pages 427-436 (1987); Tokunaga et al., "Influence of Chronic Intake of New Sweetener Fructooligosaccharide (Neosugar) on growth and Gastrointestinal Function of the Rat", JOURNAL OF NUTRITIONAL SCIENCE AND VITAMINOLOGY, Vol. 32, pages 111-121 (1986).

DEPR:
Analysis of acetate, propionate and butyrate was conducted according to Merchen et al., "Effect of Intake and Forage Level on Ruminal and Turnover Rates, Bacterial Protein Synthesis and Duodenal Amino Acid Flows in Sheep", JOURNAL OF ANIMAL SCIENCE, Vol. 62, pages 216-225 (1986). Briefly, an aliquot from the balch tube was acidified with 6N HCl and centrifuged at 31,000 .times.g for 20 minutes Concentrations of acetate, propionate and butyrate were determined in the supernatant using a Hewlett-Packard 5890A gas chromatograph and a column (180 cm.times.4 mm id) packed with 20% Tween 80-2% H.sub.3 PO.sub.4 on 60 to 80 mesh Chromosorb W (Supelco Inc, Bellefonte, Pa., U.S.A.). Nitrogen was used as a carrier gas with a flow rate of 70 mL/minutes. Oven temperature was 120.degree. C. and detector and injector temperatures were 200.degree. C. Lactate was determined colorimetrically using a method described in Barker et al., "The Colorimetric Determination of Lactic Acid in Biological Material", JOURNAL OF BIOLOGICAL CHEMISTRY, Vol. 138, page 535, (1941).

DEPR:
SCFA production (acetate, propionate, butyrate and lactate) during in vitro fermentation of the oligosaccharides is presented in Table 3. Four time points

were studied and include 3, 6, 12 and 24 hours. Retention time in the large bowel of humans will dictate the length of fermentation in vivo. In cases where retention time is great, the extent of substrate fermentability will be a factor which most influences SCFA production. If retention time is short, the rate of substrate fermentation becomes more important. Since retention times can differ significantly in an in vivo situation it is necessary to monitor substrate degradation over time in vitro in order that comparisons can be made.

DEPR:
Fermentation of all oligosaccharides was rapid, essentially being complete by 6 hours for the fructooligosaccharides (FOS and Raftilose) and by 12 hours for XOS. The results are presented in Table 5. It is recommended that the 6 hour and 12 hour values be used to estimate the composition of the end-products for the fructooligosaccharides and the XOS, respectively, even though retention times in the large bowel can be considerably longer. At later time points it becomes apparent that lactate is being converted to propionate and acetate to butyrate. Interconversion in a closed in vitro system can be a problem with rapidly fermented substrates. It does not reflect the true state of the large bowel where the fatty acids are continually absorbed.

DEPR:
As is typically found with in vitro fermentations using human fecal inoculum or in analysis of fecal samples, acetate was the short chain fatty acid found in the highest concentration. Titgemeyer et al., "Fermentability of Various Fiber Sources by Human Fecal Bacteria In Vitro", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 53, Pages 1418-1424, (1991). Baldwin., "Energy Metabolism in Anaerobes", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 23, No. 11, pages 1508-1513, (1970). determined that acetate, propionate and butyrate account for 83% of the SCFAs produced during anaerobic fermentation by large bowel microflora, and the remaining SCFAs are distributed among isovaleric, isobutyric, valeric, lactic, formic and succinic acids. In this study, a considerable amount of lactate was found, particularly during fermentation with FOS and Raftilose. It has been documented that the oligosaccharides used in this study serve as an energy source for Bifidobacteria and that there consumption will lead to the selective growth of this organism in the GI tract. Okazaki et al., "Effects of Xylooligosaccharides on the Growth of Bifidobacteria", BIFIDOBACTERIA MICROFLORA, Vol. 9, No. 2, page 77, (1990); Mitsuoka et al., "Effects of Fructo-oligosaccharide on Intestinal Microflora", DIE NAHRUNG, Vol. 31, pages 427-436, (1987). The primary end products produced by Bifidobacteria during fermentation are acetate and lactate. Miller et al., "Fermentations by Saccharolytic Intestinal Bacteria", AMERICAN JOURNAL OF CLINICAL NUTRITION, Vol. 32, pages 164-172, (1979). The fact that these oligosaccharides serve as an energy source for the Bifidobacteria could explain the elevated levels of lactate found in this study.

DEPR:
Total short-chain fatty acid production was greater for the xylooligosaccharides (XOS) compared to the fructooligosaccharides (FOS and Raftilose). The primary factor effecting the quantity of SCFAs produced during fermentation is the fermentability of the substrate. It is assumed that the oligosaccharides are completely fermented in this system. However, the yield of SCFAs (mol) from a substrate is dependent not only on the weight of the substrate fermented but also on the average molecular weight of the oligosaccharide component sugars. One can assume that the fermentation of one monosaccharide molecule can result in either two acetate, two propionate, two lactate or one molecule of butyrate. The molecular weight of the components of the fructooligosaccharides (glucose and fructose, 180) is greater than the molecular weight of xylose (150) which is the monomeric component of XOS. Subsequently, on an equivalent weight basis, there are more moles of monosaccharide molecules with the xylooligosaccharide compared to the fructooligosaccharide. This would explain the greater production of SCFA with the XOS compared to the fructooligosaccharides. Lastly, the quantity and profile of SCFAs produced was virtually identical between the two fructooligosaccharides (Raftilose and FOS). While these fructooligosaccharides differ to some extent in their chemical composition, it is apparent that they are metabolized similarly in this in vitro fermentation system.

DEPR:
Recent evidence that the regular intake of n-3 fatty acids from fish oil inhibits neutrophil and monocyte functions suggests that n-3 fatty acids have antiinflammatory properties. Beneficial effects of marine lipids have been shown in animal models of inflammatory bowel disease. Empey et al., "Fish Oil-Enriched Diet is Mucosal Protective Against Acetic Acid-Induced Colitis in Rats", CANADIAN JOURNAL OF PHYSIOLOGY AND PHARMACOLOGY, Vol. 69, pages 480-487 (1991); Vilaseca et al., "Dietary Fish Oil Reduces Progression of Chronic Inflammatory Lesions in a Rat Model of Granulomatous Colitis", GUT, Vol. 31, page 539 (1990). In preliminary therapeutic trials, diet supplementation with fish oil has led to symptomatic improvement of patients with ulcerative colitis, and reduced ethanol-induced damage in human duodenal mucosa. Schepp et al., "Fish Oil Reduces Ethanol-Induced Damage of the Duodenal Mucosa in Humans", EUROPEAN JOURNAL OF CLINICAL INVESTIGATION, Vol. 21, pages 230-237 (1991); Lorenz et al., "Supplementation with n-3 Fatty Acids from Fish Oil in Chronic Inflammatory Bowel Disease", JOURNAL OF INTERNAL MEDICINE SUPPLEMENT, Vol. 225, pages 225-232 (1989); Hillier et al., "Incorporation of Fatty Acids from Fish Oil and Olive Oil into Colonic Mucosal Lipids and Effects Upon Eicosanoid Synthesis in Inflammatory Bowel Disease", GUT, Vol. 32, pages 1151-1155 (1991); Saloman et al., "Treatment of Ulcerative Colitis with Fish Oil N-3-w-Fatty Acid: An Open Trial", JOURNAL OF CLINICAL GASTROENTEROLOGY, Vol. 12, No. 2, pages 157-161 (1990).

DEPR:
A major limitation in investigating the pathogenic mechanisms responsible for the mucosal injury observed during chronic inflammation of the intestine and colon has been the relative paucity of relevant animal models. Two models of colitis produced in rats that have received much attention over the past few years are the acetic acid and trinitrobenzene sulfonic acid (TNBS) models. The mechanism by which acetic acid produces the diffuse colitis is thought to involve nonspecific, acid induced injury to the colonic mucosa that is followed by an acute inflammatory response. Apparently the protonated form of the acid is required to induce the colitis since neither HCl (pH 2.3) nor sodium acetate (pH 7.0) is effective in eliciting the inflammatory response. However, there is some evidence to suggest that acetic acid may promote other pathophysiological events (e.g. fluid and electrolyte secretion) using noncytotoxic concentrations of the acid.

DEPR:
Recent studies have demonstrated that the intrarectal administration of the hapten, TNBS, in the presence of a mucosal barrier breaker such as ethanol, produces an acute and possibly chronic colitis in unsensitized rats. The mechanism(s) by which buffered or unbuffered TNBS in the presence of ethanol initiates inflammation in unsensitized animals is unclear; however, it has been suggested to involve macrophage-mediated recognition and lysis of TNBS-modified autologous cells within the mucosa. However, more recent evidence suggests more complicated mechanisms. For example, the barrier breaker, ethanol, is an extremely potent pro-inflammatory solvent alone. Furthermore, it has been demonstrated that TNBS is metabolized by certain colonic enzymes and substrates to yield both pro-inflammatory and cytotoxic oxidants that could initiate colonic inflammation. Grisham et al., "Metabolism of Trinitrobenzene Sulfonic Acid by the Rat Colon Produces Reactive Oxygen Species", GASTROENTEROLOGY, Vol. 101, pages 540-547 (1991). A recent study directly compared the acetic acid and the TNBS (+ETOH) models of colitis and found that either model may be useful to study those events that occur at the time of inflammation (e.g. arachidonate metabolism, granulocyte infiltration and metabolism, etc.) or during repair. However, the use of these models of colitis may have significant limitations in understanding those immunological events that initiate the acute and chronic inflammatory episodes. For example, the inflammation and tissue injury observed in human inflammatory bowel disease is most probably a result of inappropriate immunological activation (e.g. autoimmune, infectious agent, etc.) whereas the inflammation induced by the intrarectal application of acetic acid, ethanol or ethanol plus TNBS is a response to extensive mucosal injury. Thus, the mechanisms by which inflammation (and mucosal injury) are achieved in the human

disease may be very different than those in the experimental models.

DEPR:
For these reasons, a model of acute and chronic distal colitis in rats was developed based upon a previously published method in which purified bacterial cell wall polymers (derived from Group A streptococci) are injected intramurally into the distal colon of genetically-susceptible rats. Sartor et al., "Granulomatous Entercolitis Induced by Purified Bacterial Cell Wall Fragments", GASTROENTEROLOGY, Vol. 89, pages 587-595 (1985). This model produces an acute and chronic inflammation characterized by the infiltration of large numbers of inflammatory cells, enhanced mucosal permeability, interstitial fibrosis, and mucosal thickening as well as the extraintestinal manifestations of arthritis, hepatic and splenic granulomas. Unlike most models of colitis, the inflammation induced in this model promoting mucosal and submucosal injury rather than the injury causing the inflammation.

DEPR:
In Experiment 2 the objectives were: (a) to determine whether this model of colitis responds to sulfasalazine (SAZ) and (b) to assess the effects of specially formulated enteral diets on the injury and inflammation observed in the colon, liver and spleen.

DEPR:
Total dietary intake and body weights of the control and liquid diet groups were recorded for each 24 hour period during the course of the 4 week experiment (1 week prior to the induction of colitis and 3 weeks following PG/PS or albumin injection). To assess the effects of SAZ in this model, female Lewis rats were orally administered SAZ immediately following the induction of colitis. Rats were given chow ad libitum for the duration of the four week study period. Similar measurements were assessed as described below.

DEPR:
Recent studies have suggested that nutritional supplementation in the form of enteral diets may prove useful as adjunctive or primary therapy for patients with IBD. Indeed, recent reports suggest that n-3 fatty acids from fish oil as well as the SCFA produced during the fermentation of indigestible carbohydrates may attenuate some of the pathophysiology associated with active gut inflammation. Therefore, we ascertained whether three enteral diets, one supplemented with fish oil or two different diets supplemented with two forms of indigestible carbohydrate could inhibit some of the inflammation observed in a model of chronic colitis. The results are presented in FIGS. 1-5. FIG. 1 presents colon weights of animals following the various therapies (diets). FIG. 2 presents MPO activity in colonic tissue of rats following the various therapies (diets). FIG. 3 presents liver weights of animals following the various therapies (diets). FIG. 4 presents spleen weights in the animals following the various therapies (diets). FIG. 5 presents levels in circulating plasma of nitrate and nitrite in animals following the various therapies (diets).

DETL:
TABLE 5

SHORT CHAIN FATTY ACID PRODUCTION DURING 3, 6, 12 AND 24 H in vitro FERMENTATION OF VARIOUS OLIGOSACCHARIDES SHORT CHAIN FATTY ACID.sup.a Total SUBSTRATE HOUR Acetate Propionate Butyrate Lactate SCFA.sup.b

FOS 3 1.49 .20 .23 .45 2.37 6 3.61 .54 .87 1.19 6.21 12 3.67 1.01 1.64 .54 6.86 24 3.20 1.09 2.09 .01 6.39 Raftilose .RTM. 3 1.42 .20 .27 .47 2.36 6 3.49 .53 .92 1.28 6.22 12 3.68 .98 1.70 .59 6.95 24 3.09 1.05 2.1 .01 6.30 XOS 3 1.21 .15 .13 .14 1.63 6 4.12 .58 .58 .47 5.75 12 5.90 .97 1.1 .74 8.72 24 5.53 .96 1.5 .05 8.10 Statistics SEM .13 .08 .08 .08 LSD.sup.c .37 .23 .23 .23

.sup.a Calculated as (mmol fatty acid in incubation tube minus mmol fatty acid in blank tube) divided by original substrate dry matter (DM) and expressed as mmol/g substrate DM. .sup.b Sum of acetate + propionate + butyrate + lactate and

expressed as mmol/g substrate DM. .sup.c Differences between mean values within a column greater than the specified LSD are significandy different P < .05.

ORPL:
Scheppach et al., "Effect of Butyrate Enemas on the Colonic Mucosa in Distal Ulcerative Colitis", Gastroenterology, 103:51-56 (1992).

ORPL:
Vilaseca et al., "Dietary Fish Oil Reduces Progression of Chronic Inflammatory Lesions in a Rat Model of Granulomatous Colitis", GUT, 31:539-544 (1990).

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☐ 1. Document ID: US 5733579 A

L37: Entry 1 of 1 File: USPT Mar 31, 1998

US-PAT-NO: 5733579
DOCUMENT-IDENTIFIER: US 5733579 A

TITLE: Oral rehydration solution containing indigestible oligosaccharides

DATE-ISSUED: March 31, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Wolf; Bryan Warren	Newark	OH		
Garleb; Keith Allen	Powell	OH		
Campbell; Sheila Martinson	Worthington	OH		
Meulbroek; Jonathan Allan	Grayslake	IL		
Wheeler; Keith Brian	Dublin	OH		
Walton; Joseph Edward	Westerville	OH		

US-CL-CURRENT: 424/606; 424/610, 424/663, 424/717, 514/23, 514/867

ABSTRACT:

An oral rehydration solution contains indigestible oligosaccharides. Diarrhea related dehydration requires fluid and electrolyte replacement. The primary etiology of antibiotic-associated diarrhea (also known as pseudomembranous colitis) has been recognized as Clostridium difficile. It is believed that the indigenous microflora of a healthy individual suppresses the normally present C. difficile. However, when the indigenous microflora are disrupted (e.g., during antibiotic treatment) overgrowth of C. difficile may occur causing diarrhea and colitis. Treatment of diarrhea related to C. difficile with rehydration therapy and antibiotics has proven effective, but many times relapse occurs. It has been suggested that normalization of the microflora will inhibit C. difficile relapse. Indigestible oligosaccharides have been shown to inhibit C. difficile infection.

8 Claims, 12 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 8

L37: Entry 1 of 1 File: USPT Mar 31, 1998

DOCUMENT-IDENTIFIER: US 5733579 A
TITLE: Oral rehydration solution containing indigestible oligosaccharides

DEPR:
It is becoming increasingly obvious that many of the beneficial effects of fermentable carbohydrate are mediated by short chain fatty acids (SCFA) such as acetate, propionate, and butyrate, which are produced during anaerobic fermentation in the colon. Short chain fatty acids play a key role in bowel function. The absorption of 100 mmole SCFA is associated with the absorption of 360 ml water. Casparey et al., "Bacterial fermentation of carbohydrates within the gastrointestinal tract", CLINICAL RESEARCH REVIEW, (Suppl. 1):107-177 (1981). Subsequently, the absence or reduction of SCFA in the colon could result in diarrhea. Ramakrishna et al. "Colonic dysfunction in acute diarrhea: the role of luminal short chain fatty acids", GUT, 34:1215 (1993) found that fecal output of short chain fatty acids in patients with acute diarrhea was low on the first day of illness, but increased over the next five days as the patients condition improved. Further, using an in vivo rectal dialysis technique, Ramakrishna et al. "Colonic dysfunction in acute diarrhoea: the role of luminal short chain fatty acids", GUT, 34:1215 (1993) demonstrated that lumenal SCFA could restore net water and sodium reabsorption in the rectum of patients with acute diarrhea. In vivo perfusion studies in healthy subjects have shown secretion of salt and water in the ascending colon in response to enteral feeding. Bowling et al., "Colonic secretory effect in response to enteral feeding in man", GUT, 34(suppl. 1):A54 (1993); Bowling et al., "The colonic secretory response to enteral feeding:influence of high strength diet", CLINICAL NUTRITION, 12(suppl. 2):23 (1993). Bowling et al., "Reversal by short-chain fatty acids of colonic fluid secretion induced by enteral feeding", THE LANCET, 342:1266 (1993) investigated the effect of short chain fatty acids on colonic fluid secretion induced by enteral feed. The researchers found that SCFA infusion directly into the cecum of healthy subjects reversed the fluid secretion seen in the ascending colon during enteral feeding and theorized that these findings could have implications for the management of diarrhea related to enteral feedings.

DEPR:
The use of the Syrian hamster as a model for C. difficile-colitis is widely recognized. Lust et al., "Clindamycin-Induced Enterocolitis in Hamsters", THE JOURNAL OF INFECTIOUS DISEASES, 137(4) 464-475 (1978), proposed that the enterocolitis induced in the hamster by antibiotics is a good model for investigation of the syndrome in humans. Price et al., "Morphology of experimental antibiotic-associated enterocolitis in the hamster: a model for human pseudomembranous colitis and antibiotic-associated diarrhoea", GUT, 20:467-475 (1979) set out to study the morphology of experimental antibiotic-associated pseudomembranous colitis (PMC). They noted that the hamster model has some morphological differences; however, the bacteriology and toxicology are identical to the human. They concluded that the hamster is a good model for investigating the pathogenesis of PMC and antibiotic-associated enteropathy in general. In fact, Wilson et at., "Suppression of Clostridium difficile by Normal Hamster Cecal Flora and Prevention of Antibiotic-Associated Cecitis", INFECTION AND IMMUNITY, 34(2) 626-628 (1981), noted that studies with the hamster model of antibiotic-associated colitis led to the discovery of C. difficile toxin as a major etiology of antibiotic-associated colitis in humans and to effective treatment with oral vancomycin. Wilson et al., "Population Dynamics of Ingested Clostridium difficile in the Gastrointestinal Tract of the Syrian Hamster", THE JOURNAL OF INFECTIOUS DISEASES, 151(2)355-361 (1985) noted that the best studied animal model of antibiotic-associated colitis was that of the Syrian hamster.

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☐ 1. Document ID: US 6326000 B1

L43: Entry 1 of 6 File: USPT Dec 4, 2001

US-PAT-NO: 6326000
DOCUMENT-IDENTIFIER: US 6326000 B1

TITLE: Kit with enteral dietary composition consisting of Streptococcus thermophilus, Bifidobacterium infantis and Bifidobacterium longum

DATE-ISSUED: December 4, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Cavaliere ved. Vesely; Renata Maria Anna	Milan			ITX
De Simone; Claudio	Rome			ITX

US-CL-CURRENT: 424/93.3; 424/93.44, 424/93.45, 435/975

ABSTRACT:

Enteral compositions containing Streptococcus thermophilus, Bifidobacterium infantis and Bifidobacterium longum, each at a concentration equal to or greater than 1.times.10.sup.11 CFU per gram, are useful as adjuncts for enteral formulations and as oral nutritional supplements. The compositions can be administered before, during or at the end of an enteral formulation administration. The compositions can be administered separately or mixed with the enteral formulation. The compositions can also be employed at the end of the daily administration in order to prevent the colonization of the enteral tube by the other pathogens. The compositions can also be used as supplement to any liquid, creamy or pasty foodstuff.

The present invention relates to a kit comprising two containers, one containing a foodstuff and the other containing the enteral composition consisting of Streptococcus thermophilus, Bifidobacterium infantis and Bifidobacterium longum.

8 Claims, 0 Drawing figures Exemplary Claim Number: 1

L43: Entry 1 of 6 File: USPT Dec 4, 2001

DOCUMENT-IDENTIFIER: US 6326000 B1
TITLE: Kit with enteral dietary composition consisting of Streptococcus thermophilus, Bifidobacterium infantis and Bifidobacterium longum

BSPU:
1) Production of nutrients for the colonic mucosa: acetate, butyrate, propionate, other short chain fatty acids, pyruvate, lactate, and amino acids such as arginine, cysteine and glutamine;

DEPR:
Twenty patients ranging from 24 to 61 years of age with chronic ulcerative colitis (CUC), diarrhea and who had lost at least 10% of their body weight in the past two months were recruited into the study. The histological criteria of Lockhart-Mummery and Morson were used to establish the diagnosis of CUC and to distinguish this form of colitis from Crohn's disease. All patients at the entry in the trial were submitted to colonoscopy to assess the extent of CUC. Patients were excluded from the study if they were in treatment with antibiotics or had bacterial or parasitic pathogens in their stools, a positive test for Clostridium difficile toxin, and active viral or fungal infections as well as major clinical complications, such as megacolon, perforation, or septicaemia.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 2. Document ID: US 6271362 B1

L43: Entry 2 of 6 File: USPT Aug 7, 2001

US-PAT-NO: 6271362
DOCUMENT-IDENTIFIER: US 6271362 B1

TITLE: Gene encoding IGG FC region-binding protein

DATE-ISSUED: August 7, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Morikawa; Minoru	Chiba-ken			JPX
Harada; Naoki	Saitama-ken			JPX

US-CL-CURRENT: 536/23.5; 435/252.3, 435/320.1, 435/325, 435/6, 435/69.1

ABSTRACT:

A gene encoding an IgG Fc region-binding protein (FC.gamma.BP); a recombinant vector containing this gene; host cells transformed by this recombinant vector; a process for producing a recombinant protein which is obtained by incubating these host cells; and a protein having a recombinant IgG Fc region-binding activity which is obtained by the above-mentioned process.

10 Claims, 26 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 17

L43: Entry 2 of 6 File: USPT Aug 7, 2001

DOCUMENT-IDENTIFIER: US 6271362 B1
TITLE: Gene encoding IGG FC region-binding protein

BSPR:
As will be described hereinafter, it is assumed that Fc.gamma.BP is secreted onto mucosae together with mucus and traps pathogens or viruses invading the body into the mucus to thereby facilitate the excretion of these invaders, thus participating in the mechanism of protection against infection. An autoantibody produced in excess in a mucosa suffering from inflammation activates the complement system or causes cytotoxicity by macrophages, etc., thus worsening the inflammation. It is assumed that Fc.gamma.BP blocks the Fc portion of such an autoantibody to thereby inhibit the progression of the inflammation. Because of having these functions, Fc.gamma.BP might be applicable to drugs, for example, agents for protecting infection, antiinflammatory (antiphlogistic) agents or diagnostic drugs for autoimmune diseases such as ulcerative colitis and Crohn's disease, etc.

DRPR:
FIGS. 13A-13B provide morphological photographs showing CHO cells expressing the Fc.gamma.BP fragment therein. The sample (13A) had been treated with 6.4 .mu.M of methotrexate but no sodium butyrate, while the sample (13B) had been treated with 6.4 .mu.M of methotrexate and 5 mM of sodium butyrate.

DEPR:
In order to establish a cell line capable of expressing a large mount of the Fc.gamma.BP fragment described in Example 11 in a stable state, NV11ST (i.e., the partial Fc.gamma.BP cDNA) was expressed by using an expression vector pMSXND for animal cells. The vector pMSXND is one having a metallothionein promoter, which can induce the expression of a protein with sodium butyrate, etc., as an expression promoter and being constructed in such a manner as to have dhfr gene which enables gene amplification after being integrated into chromosomal DNA.

DEPR:
5.times.10.sup.5 cells of the CHO cell line, which could stably express the Fc.gamma.BP fragment at a high yield, were transferred into a dish (diameter: 100 mm) and incubated in .alpha.-MEM medium containing 6.4 .mu.M of methotrexate and 1 mg/ml of G418. If necessary, sodium butyrate was added in such an amount as to give a final concentration of 5 mM to thereby induce the expression of the protein. After 3 days, the culture supernatant (SUPL) was recovered. Then the SUP1 was centrifuged at 100,000.times.g for 60 minutes to thereby eliminate cell pieces therefrom and the supernatant (SUP2) was recovered. The cells were suspended in 500 .mu.l of a cytolysis buffer [50 mM of Tris-HCl (pH 7.5), 150 mM of NaCl, 1 mM of EDTA, 1 mM of PMSF, 10 mM of monoiodo acetamide, 10 .mu.g/ml of approtinine, 10 .mu.g/ml of leupeptin] and subjected to ultrasonication (30 seconds.times.3 times) and centrifugation at 10,000.times.g for 10 minutes at 4.degree. C. After the completion of the centrifugation, the supernatant (LYS1) was eliminated. To the residue was added 400 .mu.l of the cytolysis buffer containing 1% of NP-40 followed by ultrasonication and centrifugation. After recovering the supernatant (LYS2), the residue was dissolved in 200 .mu.l of the cytolysis buffer containing 1% of NP-40, 0.1% of SDS and 0.5% of sodium deoxycholate followed by ultrasonication and centrifugation. After recovering the supernatant (LYS3), the residue was dissolved in 100 .mu.l of the cytolysis buffer (LYS4). As a control, use was made of solutions of colonic epithelial cells (diluted 100- to 3,200-fold).

DEPR:
After incubating in the presence of methotrexate optionally followed by the treatment with sodium butyrate, the expression yield of the Fc.gamma.BP fragment after 3 days was detected by the cell staining method with the use of the monoclonal antibodies K9/K17. Thus a cell line with a high expression yield could be isolated as shown in FIG. 13.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

☐ 3. Document ID: US 6190691 B1

L43: Entry 3 of 6 File: USPT Feb 20, 2001

US-PAT-NO: 6190691
DOCUMENT-IDENTIFIER: US 6190691 B1

TITLE: Methods for treating inflammatory conditions

DATE-ISSUED: February 20, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mak; Vivien H. W.	Menlo Park	CA		

US-CL-CURRENT: 424/449; 514/859, 514/861, 514/863, 514/886, 514/887, 604/20

ABSTRACT:

The present invention provides a number of screening methods for evaluating compounds capable of suppressing cytokine production either in vitro or in vivo. The methods generally involve stimulating the production of a cytokine in a cell, exposing a portion of the cells to a putative cytokine modulating agent and determining subsequent levels of cytokine production in the cells. Additionally, the present invention provides certain compounds identified by this method.

35 Claims, 6 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 3

L43: Entry 3 of 6 File: USPT Feb 20, 2001

DOCUMENT-IDENTIFIER: US 6190691 B1
TITLE: Methods for treating inflammatory conditions

DEPR:
Examples of stratum corneum lipid perturbants include, but are not limited to, alcohol enhancers, such as alkanols with one to sixteen carbons, benzyl alcohol, butylene glycol, diethylene glycol, glycofurol, glycerides, glycerin, glycerol, phenethyl alcohol, polypropylene glycol, polyvinyl alcohol, and phenol; amide enhancers, such as N-butyl-N-dodecylacetamide, crotamiton, N,N-dimethylformamide, N,N-dimethylacetamide, N-methyl formamide, and urea; amino acids, such as L-.alpha.-amino acids and water soluble proteins; azone and azone-like compounds, such as azacycloalkanes; essential oils, such as almond oil, amyl butyrate, apricot kernel oil, avocado oil, camphor, castor oil, 1-carvone, coconut oil, corn oil, cotton seed oil, eugenol, menthol, oil of anise, oil of clove, orange oil, peanut oil, peppermint oil, rose oil, safflower oil, sesame oil, shark liver oil (squalene), soybean oil, sunflower oil, and walnut oil; vitamins and herbs, such as aloe, allantoin, black walnut extract, chamomile extract, panthenol, papain, tocopherol, and vitamin A palmitate; waxes, such as candelilla wax, carnuba wax, ceresin wax, beeswax, lanolin wax, jojoba oil, petrolatum; mixes, such as primary esters of fractionated vegetable oil fatty acids with glycerine or propylene glycol, and interesterified medium chain triglyceride oils; fatty acids and fatty acid esters, such as amyl caproate, butyl acetate, caprylic acid, cetyl ester, diethyl sebacate, dioctyl malate, elaidic acid ethyl caprylate, ethyl glycol palmitostearate, glyceryl beheate, glucose glutamate, isobutyl acetate, laureth-4, lauric acid, malic acid, methyl caprate, mineral oil, myristic acid, oleic acid, palmitic acid, PEG fatty esters, polyoxylene sorbitan monooleate, polypropylene glycols, propylene glycols, saccharose disterate, salicylic acid, sodium citrate, stearic acid, soaps, and caproic-, caprylic-, capric-, and lauric-triglycerides; macrocylics, such as butylated hydroxyanisole, cyclopentadecanolide, cyclodextrins; phospholipid and phosphate enhancers, such as dialkylphosphates, ditetradecyl phosphate, lecithin, 2-pyrrolidone derivatives, such as alkyl pyrrolidone-5-carboxylate esters, pyroglutamic acid esters, N-methyl pyrrolidone, biodegradable soft penetration enhancers, such as dioxane derivatives and dioxolane derivatives; sulphoxide enhancers, such as dimethyl sulphoxide and decylmethyl sulphoxide; acid enhancers, such as alginic acid, sorbic acid, and succinic acid; cyclic amines; imidazolinones; imidazoles; ketones, such as acetone, dimethicone, methyl ethyl ketone, and pentanedione; lanolin derivatives, such as lanolin alcohol, PEG 16 lanolin, and acetylated lanolin; oxazolines; oxazolindinones; proline esters; pyrroles, urethanes; and surfactants, such as nonoxynols, polysorbates, polyoxylene alcohols, polyoxylene fatty acid esters, sodium lauryl sulfate, and sorbitan monostearate.

DEPR:
Patients having a definite diagnosis of Crohn's disease or ulcerative colitis (based on radiological and histologic findings) are further examined via colonoscopy and barium imaging. (+)-Verapamil is administered twice or three times daily, either orally, or other means of delivery until a therapeutic benefit is achieved. Disease activity is assessed on the basis of reported symptoms, changes in weight, and other laboratory testings. Further, (+)-verapamil can be used as adjuvant or maintenance therapy of corticosteroid and other treatment regimens.

Full | Title | Citation | Front | Review | Classification | Date | Reference
KWIC | Draw Desc | Image

☐ 4. Document ID: US 6187546 B1

L43: Entry 4 of 6 File: USPT Feb 13, 2001

US-PAT-NO: 6187546
DOCUMENT-IDENTIFIER: US 6187546 B1

TITLE: Method of isolating cells

DATE-ISSUED: February 13, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
O'Neill; Ian Kenneth	Great Shelford, Cambridgeshire CB2 5JP			GBX
Loktionov; Alexandre	Cambridge CB4 3QE			GBX

US-CL-CURRENT: 435/7.1; 34/284, 435/1.3, 435/7.23, 436/174, 436/177

ABSTRACT:

The invention provides a method of isolating cells from a faecal stool, the method comprising the steps of a) cooling the stool to a temperature below its gel freezing point, and b) removing cells from the stool whilst maintaining the stool at a temperature below its gel freezing point such that the stool remains substantially intact. The invention further provides methods of purifying cells comprising use of immunomagnetic beads and/or boric acid. Cells isolated according to the invention may be used in diagnostic tests and assay procedures for monitoring a biological or biochemical property of tissue.

21 Claims, 0 Drawing figures Exemplary Claim Number: 1

L43: Entry 4 of 6 File: USPT Feb 13, 2001

DOCUMENT-IDENTIFIER: US 6187546 B1
TITLE: Method of isolating cells

BSPR:
Preferably, the present invention comprises removal of cells from the surface of the stool. The surface layers of the stool provide a particularly rich source of exfoliated cells suitable for isolation. Typically, between one-sixth and one third of colonic epithelial cells are exfoliated per day. Without prejudice to the present invention, it is believed that some of these exfoliated cells may be envisaged as forming a "sheath" around the stool, containing a low proportion of bacterial cells and other faecal matter. It is further believed that the viability of the cells in the surface layers of the stool is facilitated by i) a tendency for the cells to agglomerate into plaques, ii) the availability of oxygen at the surface of the stool and iii) their unique ability to utilize as fuel the butyrate formed locally in high concentrations by microfloral fermentation. Oxidation of butyrate is believed to provide fuel for the cells in the absence of blood-borne nourishment following exfoliation. It has been found that isolation of cells from the surface layers of the stool provides significant advantages in yield, purity and ease of recovery over isolation of cells from homogenized stool.

BSPR:
Preferably, the stool is washed with an aqueous solution containing a short chain fatty acid or salt thereof, preferably a C.sub.1-6 fatty acid or salt thereof, preferably a salt of butyric acid, more preferably sodium butyrate. It has been found washing with such an aqueous solution leads to improved recovery of exfoliated cells.

BSPR:
By reason of their general applicability, the methods of isolating cells of the present invention are not limited to the use of cells obtained according to the present invention in the diagnosis of cancer or neoplastic processes but also includes use in an assay for any other adverse process afflicting epithelial

tissues wherein examination of exfoliated cells provides information for use in diagnosis, treatment or prevention. These conditions include but are not limited to ulcerative colitis, inflammatory conditions of the relevant epithelia, and incorporation of viral DNA into the genome of some epithelial cells. Cells obtained according to the present invention may also be used in assays to assess beneficial changes, such as within the colorectal epithelium for example deliberately induced metabolic changes by preventative agents. The beneficial changes for which the cells serve as a biomarker include, but are not limited to, alterations to proliferation, to phase I carcinogen-activating enzyme activities and to protective enzymes such as glutathione-s-transferase and NADPH quinone reductase.

DEPU:
3 mM sodium butyrate,

CLPR:
7. A method according to claim 6 wherein the stool is washed with an aqueous solution containing sodium butyrate.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw. Desc | Image

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☐ 5. Document ID: US 6077504 A

L43: Entry 5 of 6 File: USPT Jun 20, 2000

US-PAT-NO: 6077504
DOCUMENT-IDENTIFIER: US 6077504 A

TITLE: Enteral dietary compositions comprising a mixture of live lactic bacteria consisting of Streptococcus thermophilus, Bifidobacterium longum and Bifidobacterium infantis

DATE-ISSUED: June 20, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Cavaliere ved. Vesley; Renata Maria Anna	Milan			ITX
De Simone; Claudio	Ardea (Roma)			ITX

US-CL-CURRENT: 424/93.3; 424/93.44, 424/93.45

ABSTRACT:

Enteral compositions containing a mixture of live lactic bacteria consisting of Streptococcus thermophilus, Bifidobacterium longum and Bifidobacterium infantis, each at a concentration equal to or greater than 1.times.10.sup.11 CFU per gram, are useful as adjuncts for enteral formulations and as oral nutritional supplements. The compositions can be administered before, during or at the end of an enteral formulation administration. The compositions can be administered separately or mixed with the enteral formulation. The compositions can also be employed at the end of the daily administration in order to prevent the colonization of the enteral tube by the other pathogens. The compositions can also be used as supplement to any liquid, creamy or pasty foodstuff.

6 Claims, 0 Drawing figures Exemplary Claim Number: 1

L43: Entry 5 of 6 File: USPT Jun 20, 2000

DOCUMENT-IDENTIFIER: US 6077504 A
TITLE: Enteral dietary compositions comprising a mixture of live lactic bacteria consisting of Streptococcus thermophilus, Bifidobacterium longum and Bifidobacterium infantis

BSPV:
1) Production of nutrients for the colonic mucosa: acetate, butyrate, propionate, other short chain fatty acids, pyruvate, lactate, and amino acids such as arginine, cysteine and glutamine;

DEPR:
Twenty patients ranging from 24 to 61 years of age with chronic ulcerative colitis (CUC), diarrhea and who had lost at least 10% of their body weight in the past two months were recruited into the study. The histological criteria of Lockhart-Mummery and Morson were used to establish the diagnosis of CUC and to distinguish this form of colitis from Crohn's disease. All patients at the entry in the trial were submitted to colonoscopy to assess the extend of CUC. Patents were excluded from the study if they were in treatment with antibiotics or had bacterial or parasitic pathogens in their stools, a positive test for Clostridium difficile toxin, and active viral or fungal infections as well as major clinical complications, such as megacolon, perforation, or septicaemia.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 6. Document ID: US 5962477 A

L43: Entry 6 of 6 File: USPT Oct 5, 1999

US-PAT-NO: 5962477
DOCUMENT-IDENTIFIER: US 5962477 A

TITLE: Screening methods for cytokine inhibitors

DATE-ISSUED: October 5, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mak; Vivian	Menlo Park	CA		

US-CL-CURRENT: 514/327; 424/78.05

ABSTRACT:

The present invention provides a number of screening methods for evaluatiing compounds capable of suppressing cytokine production either in vitro or in vivo. The methods generally involve stimulating the production of a cytokine in a cell, exposing a portion of the cells to a putative cytokine modulating agent and determining subsequent levels of cytokine production in the cells. Additionally, the present invention provides certain compounds identified by this method.

5 Claims, 6 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 3

L43: Entry 6 of 6 File: USPT Oct 5, 1999

DOCUMENT-IDENTIFIER: US 5962477 A
TITLE: Screening methods for cytokine inhibitors

DEPR:
Examples of stratum corneum lipid perturbants include, but are not limited to, alcohol enhancers, such as alkanols with one to sixteen carbons, benzyl alcohol, butylene glycol, diethylene glycol, glycofurol, glycerides, glycerin, glycerol, phenethyl alcohol, polypropylene glycol, polyvinyl alcohol, and phenol; amide enhancers, such as N-butyl-N-dodecylacetamide, crotamiton, N,N-dimethylformamide, N,N-dimethylacetamide, N-methyl formamide, and urea; amino acids, such as L-.alpha.-amino acids and water soluble proteins; azone and azone-like compounds, such as azacycloalkanes; essential oils, such as almond oil, amyl butyrate, apricot kernel oil, avocado oil, camphor, castor oil, 1-carvone, coconut oil, corn oil, cotton seed oil, eugenol, menthol, oil of anise, oil of clove, orange oil, peanut oil, peppermint oil, rose oil, safflower oil, sesame oil, shark liver oil (squalene), soybean oil, sunflower oil, and walnut oil; vitamins and herbs, such as aloe, allantoin, black walnut extract, chamomile extract, panthenol, papain, tocopherol, and vitamin A palmitate; waxes, such as candelilla wax, carnuba wax, ceresin wax, beeswax, lanolin wax, jojoba oil, petrolatum; mixes, such as primary esters of fractionated vegetable oil fatty acids with glycerine or propylene glycol, and interesterified medium chain triglyceride oils; fatty acids and fatty acid esters, such as amyl caproate, butyl acetate, caprylic acid, cetyl ester, diethyl sebacate, dioctyl malate, elaidic acid ethyl caprylate, ethyl glycol palmitostearate, glyceryl beheate, glucose glutamate, isobutyl acetate, laureth-4, lauric acid, malic acid, methyl caprate, mineral oil, myristic acid, oleic acid, palmitic acid, PEG fatty esters, polyoxylene sorbitan monooleate, polypropylene glycols, propylene glycols, saccharose disterate, salicylic acid, sodium citrate, stearic acid, soaps, and caproic-, caprylic-, capric-, and lauric-triglycerides; macrocylics, such as butylated hydroxyanisole, cyclopentadecanolide, cyclodextrins; phospholipid and phosphate enhancers, such as dialkylphosphates, ditetradecyl phosphate, lecithin, 2-pyrrolidone derivatives, such as alkyl pyrrolidone-5-carboxylate esters, pyroglutamic acid esters, N-methyl pyrrolidone, biodegradable soft penetration enhancers, such as dioxane derivatives and dioxolane derivatives; sulphoxide enhancers, such as dimethyl sulphoxide and decylmethyl sulphoxide; acid enhancers, such as alginic acid, sorbic acid, and succinic acid; cyclic amines; imidazolinones; imidazoles; ketones, such as acetone, dimethicone, methyl ethyl ketone, and pentanedione; lanolin derivatives, such as lanolin alcohol, PEG 16 lanolin, and acetylated lanolin; oxazolines; oxazolindinones; proline esters; pyrroles, urethanes; and surfactants, such as nonoxynols, polysorbates, polyoxylene alcohols, polyoxylene fatty acid esters, sodium lauryl sulfate, and sorbitan monostearate.

DEPR:
Patients having a definite diagnosis of Crohn's disease or ulcerative colitis (based on radiological and histologic findings) are further examined via colonoscopy and barium imaging. (+)-Verapamil is administered twice or three times daily, either orally, or other means of delivery until a therapeutic benefit is achieved. Disease activity is assessed on the basis of reported symptoms, changes in weight, and other laboratory testings. Further, (+)-verapamil can be used as adjuvant or maintenance therapy of corticosteroid and other treatment regimens.

Full | Title | Citation | Front | Review | Classification | Date | Reference
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☐ 1. Document ID: US 6204051 B1

L48: Entry 1 of 6 File: USPT Mar 20, 2001

US-PAT-NO: 6204051
DOCUMENT-IDENTIFIER: US 6204051 B1

TITLE: Apparatus and method for growing anaerobic microorganisms

DATE-ISSUED: March 20, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Copeland; James C.	Ashland	OH		
Adler; Howard I.	late of Oak Ridge	TN		
Spady; Gerald E.	Oak Ridge	TN		

US-CL-CURRENT: 435/305.4; 435/288.3, 435/303.2, 435/801

ABSTRACT:

An apparatus for growing anaerobic microorganisms is provided having a dish top that contains a sealing ring upon which the media surface in the dish bottom rests when the apparatus is inverted. The contact between the sealing ring and the media surface forms a seal that traps the gas in the headspace between the media surface and the inside of the dish top. A oxygen reducing agent can also be incorporated into the media together, in some instances, with a substrate which react with oxygen in the media and with oxygen in the headspace thereby creating an environment suitable for growing anaerobic, microaerophilic and facultative anaerobic microorganisms.

20 Claims, 18 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 8

L48: Entry 1 of 6 File: USPT Mar 20, 2001

DOCUMENT-IDENTIFIER: US 6204051 B1
TITLE: Apparatus and method for growing anaerobic microorganisms

DEPR:
The dish bottom 12 can be of any convenient dimension, and is usually circular so that this dimension is referenced as a diameter. Typically, the diameter of the dish bottom is about eight (8.0) to fifteen (15.0) cm. The depth of the dish bottom 12 defined by the height of the side wall as it extends upwardly from the base can vary and is generally about 0.8 to 1.8 cm. In certain embodiments (FIG. 4C), the base 22 of the dish bottom 12 can be divided into two, three, four or more sections 38 by sectional dividers, grid markings or other indicia 40 to enhance differential diagnostics of microorganisms (FIG. 4C).

DEPV:
Clostridium tertium, C. difficile, C. perfringens, C. cadaveris, C. acetobutylicum, Bacteroides thetaiotaomicron, B. fragilis, B. distasonis, Escherichia coli, Fusobacterium varium, F. mortiferum, F. necrophorum, Peptostreptococcus magnus, P. anaerobius, P. nigra, P. intermedius, Lactobacillus casei, L. acidophilus, Eubacterium lentuum, Bifidobacterium breve, and Streptococcus fecalis.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

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☐ 2. Document ID: US 5955344 A

L48: Entry 2 of 6 File: USPT Sep 21, 1999

US-PAT-NO: 5955344
DOCUMENT-IDENTIFIER: US 5955344 A

TITLE: Apparatus and method for growing anaerobic microorganisms

DATE-ISSUED: September 21, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Copeland; James C.	Ashland	OH		
Adler; Howard I.	Oak Ridge	TN		
Spady; Gerald E.	Oak Ridge	TN		

US-CL-CURRENT: 435/243; 435/288.3, 435/303.2, 435/305.4, 435/307.1, 435/395, 435/420, 435/801

ABSTRACT:

An apparatus for growing anaerobic microorganisms is provided having a dish top that contains a sealing ring upon which the media surface in the dish bottom rests when the apparatus is inverted. The contact between the sealing ring and the media surface forms a seal that traps the gas in the headspace between the media surface and the inside of the dish top. A oxygen reducing agent can also be incorporated into the media together, in some instances, with a substrate which react with oxygen in the media and with oxygen in the headspace thereby creating an environment suitable for growing anaerobic, microaerophilic and facultative anaerobic microorganisms.

30 Claims, 15 Drawing figures Exemplary Claim Number: 12
Number of Drawing Sheets: 6

L48: Entry 2 of 6 File: USPT Sep 21, 1999

DOCUMENT-IDENTIFIER: US 5955344 A
TITLE: Apparatus and method for growing anaerobic microorganisms

DEPR:
The dish bottom 12 can be of any convenient dimension, and is usually circular so that this dimension is referenced as a diameter. Typically, the diameter of the dish bottom is about eight (8.0) to fifteen (15.0) cm. The depth of the dish bottom 12 defined by the height of the side wall as it extends upwardly from the base can vary and is generally about 0.8 to 1.8 cm. In certain embodiments (FIG. 4C), the base 22 of the dish bottom 12 can be divided into two, three, four or more sections 38 by sectional dividers, grid markings or other indicia 40 to enhance differential diagnostics of microorganisms (FIG. 4C).

DEPR:
Using this technique with the culture dish, i.e., "OxyDish.TM." and a biocatalytic oxygen reducing agent, the following microorganisms have been grown: Clostridium tertium, C. difficile, C. perfringens, C. cadaveris, C. acetobutylicum, Bacteroides thetaiotaomicron, B. fragilis, B. distasonis, Escherichia coli, Fusobacterium varium, F. mortiferum, F. necrophorum, Peptostreptococcus magnus, P. anaerobius, P. nigra, P. intermedius, Lactobacillus casei, L. acidophilus, Eubacterium lentum, Bifidobacterium breve, and Streptococcus fecalis.

Full | Title | Citation | Front | Review | Classification | Date | Reference
KWIC | Draw. Desc | Image

☐ 3. Document ID: US 5830746 A

L48: Entry 3 of 6 File: USPT Nov 3, 1998

US-PAT-NO: 5830746
DOCUMENT-IDENTIFIER: US 5830746 A

TITLE: Apparatus and method for growing anaerobic microorganisms

DATE-ISSUED: November 3, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Copeland; James C.	Ashland	OH		
Adler; Howard I.	Oak Ridge	TN		
Spady; Gerald E.	Oak Ridge	TN		

US-CL-CURRENT: 435/243; 435/303.2, 435/305.4

ABSTRACT:

An apparatus for growing anaerobic microorganisms is provided having a dish top that contains a sealing ring upon which the media surface in the dish bottom rests when the apparatus is inverted. The contact between the sealing ring and the media surface forms a seal that traps the gas in the headspace between the media surface and the inside of the dish top. A oxygen reducing agent can also be incorporated into the media together, in some instances, with a substrate which react with oxygen in the media and with oxygen in the headspace thereby creating an environment suitable for growing anaerobic, microaerophilic and facultative anaerobic microorganisms.

22 Claims, 15 Drawing figures Exemplary Claim Number: 22
Number of Drawing Sheets: 6

L48: Entry 3 of 6 File: USPT Nov 3, 1998

DOCUMENT-IDENTIFIER: US 5830746 A
TITLE: Apparatus and method for growing anaerobic microorganisms

DEPR:
The dish bottom 12 can be of any convenient dimension, and is usually circular so that this dimension is referenced as a diameter. Typically, the diameter of the dish bottom is about eight (8.0) to fifteen (15.0) cm. The depth of the dish bottom 12 defined by the height of the side wall as it extends upwardly from the base can vary and is generally about 0.8 to 1.8 cm. In certain embodiments (FIG. 4C), the base 22 of the dish bottom 12 can be divided into two, three, four or more sections 38 by sectional dividers, grid markings or other indicia 40 to enhance differential diagnostics of microorganisms (FIG. 4C).

DEPR:
Using this technique with the culture dish, i.e., "OxyDish.TM." and a biocatalytic oxygen reducing agent, the following microorganisms have been grown: Clostridium tertium, C. difficile, C. perfringens, C. cadaveris, C. acetobutylicum, Bacteroides thetaiotaomicron, B. fragilis, B. distasonis, Escherichia coli, Fusobacterium varium, F. mortiferum, F. necrophorum, Peptostreptococcus magnus, P. anaerobius, P. nigra, P. intermedius, Lactobacillus casei, L. acidophilus, Eubacterium lentum, Bifidobacterium breve, and Streptococcus fecalis.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 4. Document ID: US 4235964 A

L48: Entry 4 of 6 File: USPT Nov 25, 1980

US-PAT-NO: 4235964
DOCUMENT-IDENTIFIER: US 4235964 A

TITLE: Method for testing and identifying microorganisms

DATE-ISSUED: November 25, 1980

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bochner; Barry R.	Alameda	CA	94501	

US-CL-CURRENT: 435/34; 435/38

ABSTRACT:

In accordance with the invention there is provided a method for identifying or testing an anaerobic microorganism wherein an aqueous suspension of a culture of the microorganism having a culture density of about 2.times.10.sup.8 cells/ml, is brought into contact with an aqueous solution containing an oxidation-reduction indicator which substantially irreversibly undergoes a change in color upon being reduced and a biodegradable test substrate compound which, when catabolized by a microorganism, will engender reduction of the indicator, said aqueous suspension containing nutrient in a concentration sufficient to support microorganism culture growth without engendering reduction of the indicator when said suspension contacts said solution, and said suspension also containing a compound which reduces O.sub.2 but does not reduce the indicator.

3 Claims, 4 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 1

L48: Entry 4 of 6 File: USPT Nov 25, 1980

DOCUMENT-IDENTIFIER: US 4235964 A
TITLE: Method for testing and identifying microorganisms

DEPR:
One of the classes of microorganisms quite commonly encountered in medical diagnostic, treatment and research work is Salmonella typhimurium. Also is well known to those in the field of microbiology, there are numerous strains of Salmonella typhimurium and considerable work has already been done and is reported in the literature with respect to the catabolic behavior of each of a number of the strains. For purposes of this example of the practice of the present invention it will be assumed that the specimen to be tested and identified contains one or the other of six different strains or species of Salmonella typhimurium which will here simply be designated as Strains 1 through 6.

DEPR:
I have now used this method to test B. fragilis, B. thetaiotaomicron, C. perfringens, and F. varium. The test results I have obtained are substantially in agreement with those obtained using conventional methods, (i.e. a pH indicator); but with my test method providing greater speed and sensitivity and not being limited only to the use of test substrate compounds which affect pH when catabolized.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

☐ 5. Document ID: US 4181574 A

L48: Entry 5 of 6 File: USPT Jan 1, 1980

US-PAT-NO: 4181574
DOCUMENT-IDENTIFIER: US 4181574 A

TITLE: Production of antibiotics by fermentation of novel strains of Micropolyspora caesia

DATE-ISSUED: January 1, 1980

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kawaguchi; Hiroshi	Tokyo			JPX
Tsukiura; Hiroshi	Mitaka			JPX
Tomita; Koji	Kawasaki			JPX

US-CL-CURRENT: 435/77; 435/119, 435/822, 435/85

ABSTRACT:

A novel antibiotic complex designated herein as Bu-2313 is produced by fermentation of Micropolyspora sp. A.T.C.C. 31295, 31296, 31297 and 31298. The complex is separated into two bioactive components, Bu-2313A and Bu-2313B, which are structurally related to the streptolydigin-tirandamycin group of antibiotics. They are active against anaerobic bacteria.

7 Claims, 6 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 6

L48: Entry 5 of 6 File: USPT Jan 1, 1980

DOCUMENT-IDENTIFIER: US 4181574 A
TITLE: Production of antibiotics by fermentation of novel strains of Micropolyspora caesia

DEPR:
Strain E864-61 contains meso-DAP, galactose and rhamnose as diagnostic cell-wall components. The composition of the cell wall is shown in Table 2.

DETL:
Table 7

In vitro Activity Against Anaerobic Bacteria MIC (mcg/ml) Code No. Test organism	Bu-2313A	Bu-2313B	Tirandamycin	Clindamycin
Bf-1 Bacteroides fragilis A20926	0.2	0.1	0.2	0.05
Bf-3 Bacteroides fragilis A20928-1	0.1	0.1	0.1	0.05
Bf-4 Bacteroides fragilis A20929	0.2	0.1	0.2	0.05
Bf-6 Bacteroides fragilis A20930	0.2	0.1	0.1	0.05
Bf-7 Bacteroides fragilis A20932	0.2	0.1	0.1	0.05
Bf-10 Bacteroides fragilis A20935	0.2	0.1	0.1	0.025
Sm-1 Sphaerophorus necrophorus A15202	0.2	0.1	0.1	0.025
So-1 Sphaerophorus pseudonecrophorus A20013	0.2	0.1	0.2	0.05
Fo-1 Fusobacterium mortiferum ATCC 9817	0.2	0.1	0.1	0.05
Fv-1 Fusobacterium varium ATCC 8501	0.1	0.1	0.1	0.05
Ae-1 Acidoaminococcus fermentans ATCC 25085	0.2	0.1	0.1	0.05
Vp-1 Veillonella parvula ATCC 17745	0.2	0.1	0.1	0.05
Cb-1 Clostridium acetobutylicum IAM 19011	0.4	0.2	0.2	0.1
Cc-1 Clostridium caproicum IAM 19228	0.4	0.2	0.2	0.1
Ch-1 Clostridium chavoei A9561	0.2	0.2	0.2	0.025
Cp-1 Clostridium perfringens A9635	0.4	0.2	0.1	0.025
Cp-2 Clostridium perfringens A21284	0.4	0.2	0.1	0.025
Pp-1 Peptocuccus prevotii ATCC 9321	0.2	0.1	0.2	0.1
Pe-101 Peptococcus aerogenes ATCC 14963	0.2	0.2	0.1	0.2
Pb-1 Peptostreptococcus anaerobius B43	0.4	0.4	0.2	0.8

☐ 6. Document ID: US 4169096 A

L48: Entry 6 of 6 File: USPT Sep 25, 1979

US-PAT-NO: 4169096
DOCUMENT-IDENTIFIER: US 4169096 A

TITLE: Antibiotic compounds

DATE-ISSUED: September 25, 1979

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kawaguchi; Hiroshi	Tokyo			JPX
Tsukiura; Hiroshi	Mitaka			JPX
Tomita; Koji	Kawasaki			JPX

US-CL-CURRENT: 548/526

ABSTRACT:

A novel antibiotic complex designated herein as Bu-2313 is produced by fermentation of Micropolyspora sp. A.T.C.C. 31295, 31296, 31297 and 31298. The complex is separated into two bioactive components, Bu-2313A and BU-2313B, which are structurally related to the streptolydigin-tirandamycin group of antibiotics. They are active against anaerobic bacteria.

5 Claims, 6 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 6

L48: Entry 6 of 6 File: USPT Sep 25, 1979

DOCUMENT-IDENTIFIER: US 4169096 A
TITLE: Antibiotic compounds

DEPR:
Strain E864-61 contains meso-DAP, galactose and rhamnose as diagnostic cell-wall components. The composition of the cell wall is shown in Table 2.

DETL:
Table 7

In vitro Activity Against Anaerobic Bacteria MIC (mcg/ml) Code No. Test organism Bu-2313A Bu-2313B Tirandamycin Clindamycin

Bf-1 Bacteroides fragilis A20926 0.2 0.1 0.2 0.05 Bf-3 Bacteroides fragilis A20928-1 0.1 0.1 0.1 0.05 Bf-4 Bacteroides fragilis A20929 0.2 0.1 0.2 0.05 Bf-6 Bacteroides fragilis A20930 0.2 0.1 0.1 0.05 Bf-7 Bacteroides fragilis A20932 0.2 0.1 0.1 0.05 Bf-10 Bacteroides fragilis A20935 0.2 0.1 0.1 0.025 Sm-1 Sphaerophorus necrophorus A15202 0.2 0.1 0.1 0.025 So-1 Sphaerophorus pseudonecrophorus A20013 0.2 0.1 0.2 0.05 Fo-1 Fusobacterium mortiferum ATCC 9817 0.2 0.1 0.1 0.05 Fv-1 Fusobacterium varium ATCC 8501 0.1 0.1 0.1 0.05 Ae-1 Acidoaminococcus fermentans ATCC 25085 0.2 0.1 0.1 0.05 Vp-1 Veillonella parvula ATCC 17745 0.2 0.1 0.1 0.05 Cb-1 Clostridium acetobutylicum IAM 19011 0.4 0.2 0.2 0.1 Cc-1 Clostridium caproicum IAM 19228 0.4 0.2 0.2 0.1 Ch-1 Clostridium chavoei A9561 0.2 0.2 0.2 0.025 Cp-1 Clostridium perfringens A9635 0.4 0.2 0.1 0.025 Cp-2 Clostridium perfringens A21284 0.4 0.2 0.1 0.025 Pp-1 Peptocuccus prevotii ATCC 9321 0.2 0.1 0.2 0.1 Pe-101 Peptococcus aerogenes ATCC 14963 0.2 0.2 0.1 0.2 Pb-1 Peptostreptococcus anaerobius B43 0.4 0.4 0.2 0.8

Full | Title | Citation | Front | Review | Classification | Date | Reference
KWIC | Draw Desc | Image

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☐ 1. Document ID: US 20010028885 A1

L52: Entry 1 of 10 File: PGPB Oct 11, 2001

PGPUB-DOCUMENT-NUMBER: 20010028885
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20010028885 A1

TITLE: Bovine footrot treatment and prevention

PUBLICATION-DATE: October 11, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Morck, Douglas W.	Airdrie		CA	
Olson, Merle E.	Calgary		CA	

US-CL-CURRENT: 424/236.1; 435/252.1

ABSTRACT:

This invention provides compositions and methods for treating or preventing footrot, in particular bovine footrot, by administering Porphyromonas and/or Prevotella and/or subunits and/or toxins thereof or neutralizing agents such as antibodies thereto. A model useful for evaluating the effectiveness of footrot treatments or preventatives is also provided.

L52: Entry 1 of 10 File: PGPB Oct 11, 2001

DOCUMENT-IDENTIFIER: US 20010028885 A1
TITLE: Bovine footrot treatment and prevention

BSTX:
[0013] Our isolation techniques and our immunology studies suggest that Porphyromonas (and especially P. levii) and Prevotella (and especially P. intermedia) are more appropriate vaccine candidates than Fusobacterium necrophorum. Using surgical biopsy techniques and stringent anaerobic culture methodology, we have not isolated Fusobacterium necrophorum from internal infected tissues in a single case of bovine footrot.

DETX:
[0187] 8. Nagaraja, et al., Fusobacterium necrophorum Leukotoxoid Vaccine, U.S. Pat. No. 5,455,034 (1995).

DETX:
[0188] 9. Nagaraja, et al., Fusobacterium Leukotoxoid Vaccine, U.S. Pat. No. 5,492,694 (1996).

2. Document ID: US 6287575 B1

L52: Entry 2 of 10 File: USPT Sep 11, 2001

US-PAT-NO: 6287575
DOCUMENT-IDENTIFIER: US 6287575 B1

TITLE: Vaccine against papillomatous digital dermatitis (PDD)

DATE-ISSUED: September 11, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Walker; Richard L.	Davis	CA		
Read; Deryck H.	Yucaipa	CA		
Hird; David W.	Davis	CA		
Lefebvre; Rance B.	Davis	CA		
Berry; Steven L.	Davis	CA		
Cullor; James S.	Woodland	CA		
Lefler; Hank M.	Reno	NV		

US-CL-CURRENT: 424/262.1; 424/184.1, 424/234.1, 424/823, 424/93.1, 424/93.4, 435/243, 435/252.1

ABSTRACT:

This invention relates to the diagnosis and prevention of ungulate diseases caused by the spirochete bacteria Treponema. The invention specifically relates to isolated cultures of this spirochete and isolated nucleic acids and proteins.

7 Claims, 0 Drawing figures Exemplary Claim Number: 1

L52: Entry 2 of 10 File: USPT Sep 11, 2001

DOCUMENT-IDENTIFIER: US 6287575 B1
TITLE: Vaccine against papillomatous digital dermatitis (PDD)

BSPR:
In addition to the Treponema antigen, the vaccine can also include antigens to other ungulate diseases. For example, the vaccine can include antigens to ungulate Fusobacterium necrophorum, Porphyromonas levii, and Dichelobacter nodosus (the organisms that cause interdigital necrobacillosis, commonly known as foot rot), leptospiral bacteria, bovine respiratory syncytial virus, bovine Herpes virus, bovine diarhhea virus, bovine parainfluenza virus, vesicular stomatitis virus, malignant catarrhal fever virus, blue tongue virus, pseudorabies virus, rabies virus, rinderpest virus, and Clostridia spp. antigen.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

3. Document ID: US 6241992 B1

L52: Entry 3 of 10 File: USPT Jun 5, 2001

US-PAT-NO: 6241992
DOCUMENT-IDENTIFIER: US 6241992 B1

TITLE: Bovine footrot treatment and prevention

DATE-ISSUED: June 5, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Morck; Douglas W.	Airdrie			CAX
Olson; Merle E.	Calgary			CAX

US-CL-CURRENT: 424/236.1; 424/130.1, 424/184.1, 424/234.1, 424/278.1, 424/282.1, 424/823, 435/23, 435/30, 435/69.3, 435/7.32, 435/71.2, 435/71.3, 514/12, 514/2

ABSTRACT:

This invention provides compositions and methods for treating or preventing footrot, in particular bovine footrot, by administering Porphyromonas and/or Prevotella and/or subunits and/or toxins thereof or neutralizing agents such as antibodies thereto. A model useful for evaluating the effectiveness of footrot treatments or preventatives is also provided.

10 Claims, 21 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 11

L52: Entry 3 of 10 File: USPT Jun 5, 2001

DOCUMENT-IDENTIFIER: US 6241992 B1
TITLE: Bovine footrot treatment and prevention

BSPR:
Our isolation techniques and our immunology studies suggest that Porphyromonas (and especially P. levii) and Prevotella (and especially P. intermedia) are more appropriate vaccine candidates than Fusobacterium necrophorum. Using surgical biopsy techniques and stringent anaerobic culture methodology, we have not isolated Fusobacterium necrophorum from internal infected tissues in a single case of bovine footrot.

DEPU:
8. Nagaraja, et al., Fusobacterium necrophorum Leukotoxoid Vaccine, U.S. Pat. No. 5,455,034 (1995).

DEPU:
9. Nagaraja, et al., Fusobacterium Leukotoxoid Vaccine, U.S. Pat. No. 5,492,694 (1996).

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw. Desc | Image

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☐ 4. Document ID: US 6132709 A

L52: Entry 4 of 10 File: USPT Oct 17, 2000

US-PAT-NO: 6132709
DOCUMENT-IDENTIFIER: US 6132709 A

TITLE: Bacterin for the treatment of necrophorum diseases and a method for the production thereof

DATE-ISSUED: October 17, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Berg; John N.	Columbia	MO		

US-CL-CURRENT: 424/93.4; 435/243, 435/245, 435/252.1

ABSTRACT:

A method for treating cattle and sheep to prevent foot rot and/or liver necrosis comprising administering a Fusobacterium necrophorum bacterin which is a .beta.-propriolactone inactivated Fusobacterium necrophorum isolate to the animal being treated.

10 Claims, 0 Drawing figures Exemplary Claim Number: 1

L52: Entry 4 of 10 File: USPT Oct 17, 2000

DOCUMENT-IDENTIFIER: US 6132709 A
TITLE: Bacterin for the treatment of necrophorum diseases and a method for the production thereof

BSPR:
Abe et al discloses in "Immunization of Mice Against Fusobacterium necrophorum Infection by Parenteral or Oral Administration of Vaccine", Am. J. Vet. Res., Vol. 39, No. 1, pages 115-118 (January 1978) a vaccine made with whole cell suspensions of formalin-killed F. necrophorum. This vaccine was administered by three different routes: intraperitoneal injection of the killed cells in a saline solution, intraperitoneal injection of the killed cells with added aluminum hydroxide adjuvant and by feeding as a powder to which lyophilized bacterial cells had been added. However, even the most effective treatment (i.e., IP injection of cells plus adjuvant) resulted in mortality rates of almost 40% after seven days post challenge. These bacterins have not, however, shown sufficient efficacy when tested under field conditions to be of commercial value.

DEPR:
Results of these studies showed a significant reduction in fusobacterium disease among vaccinated sheep and demonstrated the efficacy of BPL inactivated bacterin under field conditions.

CLPR:
9. A method for treating cattle and sheep to prevent foot rot or liver necrosis comprising administering a Fusobacterium necrophorum vaccine comprising a .beta.-propriolactone inactivated Fusobacterium necrophorum isolate and an adjuvant, to the animal being treated.

ORPL:
Abe et al, "Immunization of Mice Against Fusobacterium necrophorum Infection by Parenteral or Oral Administration of Vaccine", Am. J. Vet. Res., vol. 39, No. 1, pp. 115-118, Jan., 1978.

☐ 5. Document ID: US 6096323 A

L52: Entry 5 of 10 File: USPT Aug 1, 2000

US-PAT-NO: 6096323
DOCUMENT-IDENTIFIER: US 6096323 A

TITLE: Vaccine against papillomatous digital dermatitis (PDD)

DATE-ISSUED: August 1, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Walker; Richard L.	Davis	CA		
Read; Deryck H.	Yucaipa	CA		
Hird; David W.	Davis	CA		
Lefebvre; Rance B.	Davis	CA		
Berry; Steven L.	Davis	CA		
Cullor; James S.	Woodland	CA		
Lefler; Hank M.	Reno	NV		

US-CL-CURRENT: 424/262.1; 424/184.1, 424/234.1, 424/823, 424/93.1, 424/93.4, 435/243, 435/252.1

ABSTRACT:

This invention relates to the diagnosis and prevention of ungulate diseases caused by the spirochete bacteria Treponema. The invention specifically relates to isolated cultures of this spirochete and isolated nucleic acids and proteins.

8 Claims, 1 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 1

L52: Entry 5 of 10 File: USPT Aug 1, 2000

DOCUMENT-IDENTIFIER: US 6096323 A
TITLE: Vaccine against papillomatous digital dermatitis (PDD)

DEPR:
In addition to the Treponema antigen, the vaccine can also include antigens to other ungulate diseases. For example, the vaccine can include antigens to ungulate Fusobacterium necrophorum, Porphyromonas levii, and Dichelobacter nodosus (the organisms that cause interdigital necrobacillosis, commonly known as foot rot), leptospiral bacteria, bovine respiratory syncytial virus, bovine Herpes virus, bovine diarhhea virus, bovine parainfluenza virus, vesicular stomatitis virus, malignant catarrhal fever virus, blue tongue virus, pseudorabies virus, rabies virus, rinderpest virus, and Clostridia spp. antigen.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 6. Document ID: US 5492694 A

L52: Entry 6 of 10 File: USPT Feb 20, 1996

US-PAT-NO: 5492694
DOCUMENT-IDENTIFIER: US 5492694 A

TITLE: Fusobacterium leukotoxoid vaccine

DATE-ISSUED: February 20, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Nagaraja; Tiruvoor G.	Manhattan	KS		
Chengappa; Muckatira M.	Manhattan	KS		

US-CL-CURRENT: 424/236.1; 424/130.1, 424/164.1, 424/176.1, 424/184.1, 424/197.11, 435/243, 435/252.1, 435/71.3, 435/822

ABSTRACT:

A method is provided for the enhanced elaboration of leukotoxin from F. necrophorum, and subsequent production of an inactivated leukotoxoid ruminant animal vaccine against F. necrophorum infection and consequent liver abscesses and/or foot rot in such animals. The method involves forming a culture of F. necrophorum bacteria in growth media, allowing the bacteria to grow therein and to simultaneously elaborate leukotoxin in a supernate; the culturing is preferably carried out at a temperature of from about 35.degree.-41.degree. C., a pH of from about 6.5-8, and for a period of from about 4-9 hours. At the end of the culturing, bacterial growth and leukotoxin elaboration are terminated, preferably by separating the leukotoxin supernate, whereupon the vaccine is produced by inactivation of at least the supernate.

24 Claims, 7 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 3

L52: Entry 6 of 10 File: USPT Feb 20, 1996

DOCUMENT-IDENTIFIER: US 5492694 A
TITLE: Fusobacterium leukotoxoid vaccine

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

☐ 7. Document ID: US 5455034 A

L52: Entry 7 of 10 File: USPT Oct 3, 1995

US-PAT-NO: 5455034
DOCUMENT-IDENTIFIER: US 5455034 A

TITLE: Fusobacterium necrophorum leukotoxoid vaccine

DATE-ISSUED: October 3, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Nagaraja; Tiruvoor G.	Manhattan	KS		
Chengappa; Muckatira M.	Manhattan	KS		

US-CL-CURRENT: 424/236.1; 424/130.1, 424/164.1, 424/176.1, 424/184.1, 424/197.11, 435/243, 435/252.1, 435/71.3, 435/822

ABSTRACT:

A method is provided for the enhanced elaboration of leukotoxin from F. necrophorum, and subsequent production of an inactivated leukotoxoid ruminant animal vaccine against F. necrophorum infection and consequent liver abscesses and/or foot rot in such animals. The method involves forming a culture of F. necrophorum bacteria in growth media, allowing the bacteria to grow therein and to simultaneously elaborate leukotoxin in a supernate; the culturing is preferably carried out at a temperature of from about 35.degree.-41.degree. C., a pH of from about 6.5-8, and for a period of from about 4-9 hours. At the end oil of the culturing, bacterial growth and leukotoxin elaboration are terminated, preferably by separating the leukotoxin supernate, whereupon the vaccine is produced by inactivation of at least the supernate.

3 Claims, 7 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 3

L52: Entry 7 of 10 File: USPT Oct 3, 1995

DOCUMENT-IDENTIFIER: US 5455034 A
TITLE: Fusobacterium necrophorum leukotoxoid vaccine

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw. Desc | Image

☐ 8. Document ID: US 4203968 A

L52: Entry 8 of 10 File: USPT May 20, 1980

US-PAT-NO: 4203968
DOCUMENT-IDENTIFIER: US 4203968 A

TITLE: Combination vaccine for swine dysentery and method of use

DATE-ISSUED: May 20, 1980

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Harris; Delbert L.	Ames	IA		
Goodnow; Robert A.	Omaha	NE		
Glock; Robert D.	Ames	IA		
Kinyon; Joann M.	Ames	IA		

US-CL-CURRENT: 424/203.1; 424/234.1, 424/262.1, 424/825

ABSTRACT:

A combination vaccine for increasing the resistance of swine to dysentery infection comprises killed cells of a virulent isolate of Treponema hyodysenteriae in combination with concentrated killed cells of Bacteroides vulgatus, or Fusobacterium necrophorum, or mixtures thereof. This combination vaccine can be adapted for either oral or parenteral administration. For parenteral administration preferably only Bacteroides vulgatus is used in combination with Treponema hyodysenteriae. The oral vaccine is enteric-coated.

5 Claims, 0 Drawing figures Exemplary Claim Number: 1

L52: Entry 8 of 10 File: USPT May 20, 1980

DOCUMENT-IDENTIFIER: US 4203968 A
TITLE: Combination vaccine for swine dysentery and method of use

ABPL:
A combination vaccine for increasing the resistance of swine to dysentery infection comprises killed cells of a virulent isolate of Treponema hyodysenteriae in combination with concentrated killed cells of Bacteroides vulgatus, or Fusobacterium necrophorum, or mixtures thereof. This combination vaccine can be adapted for either oral or parenteral administration. For parenteral administration preferably only Bacteroides vulgatus is used in combination with Treponema hyodysenteriae. The oral vaccine is enteric-coated.

BSPR:
For use as an oral vaccine, Fusobacterium necrophorum in the form of concentrated killed cells can be employed instead of or in addition to Bacteroides vulgatus. One preferred oral vaccine contains both Bacteroides vulgatus and Fusobacterium necrophorum cells with the T. hyodysenteriae antigen, and each are used in the relative proportions to T. hyodysenteriae specified above with respect to B. vulgatus.

BSPR:
For preparing vaccines in accordance with the present invention, it is believed that any strain or isolate of Bacteroides vulgatus or Fusobacterium necrophorum can be used. Strains of these bacteria which can be used in practicing the present invention are therefore readily available, such as those on deposit with the American Type Culture Collection, Rockville Maryland. For B. vulgatus these include the type strain ATCC No. 8482, and other strains or isolates, such as ATCC No. 31376.

☐ 9. Document ID: US 4152415 A

L52: Entry 9 of 10 File: USPT May 1, 1979

US-PAT-NO: 4152415
DOCUMENT-IDENTIFIER: US 4152415 A

TITLE: Method of increasing the effectiveness of oral vaccination for swine dysentery

DATE-ISSUED: May 1, 1979

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Harris; Delbert L.	Ames	IA		
Goodnow; Robert A.	Omaha	NE		

US-CL-CURRENT: 424/494; 424/262.1, 424/498, 424/825

ABSTRACT:

The resistance of field-raised swine to dysentery infection is increased by a sequence of parenteral and oral administrations. First there is parenterally administered to the swine an injectable cell concentrate containing a virulent isolate of killed cells of Treponema hyodysenteriae, and not less than five days after the animals have received the first of the parenteral injections, a series of oral administrations of enteric-coated pellets is started, the pellets containing concentrated killed cells of a virulent isolate of T. hyodysenteriae. The oral administrations are given at least once every 24 hours for a period of at least five days.

10 Claims, 0 Drawing figures Exemplary Claim Number: 1

L52: Entry 9 of 10 File: USPT May 1, 1979

DOCUMENT-IDENTIFIER: US 4152415 A
TITLE: Method of increasing the effectiveness of oral vaccination for swine dysentery

BSPR:
The method of this invention can also be practiced by using the combination vaccine for swine dysentery described in the co-pending application of Delbert L. Harris, Robert A. Goodnow, Robert D. Glock, and Joann M. Kinyon, filed on even date herewith, and entitled "Combination Vaccine for Swine Dysentery and Method of Use". More specifically, the oral vaccine may contain other bacteria in addition to T. hyodysenteriae, such as Bacteroides vulgatus, or Fusobacterium necrophorum, or mixtures thereof. For example, at least one of these other bacteria may be present in an amount of from 0.25 to 2 parts by weight (dry basis) per part of T. hyodysenteriae cells, the additional bacteria being present in the form of concentrated killed cells. The parenteral preparation may also contain concentrated killed cells of B. vulgatus in the amount of from 0.25 to 2 parts by weight (dry basis) of the B. vulgatus cells per part of the T. hyodysenteriae cells.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 10. Document ID: US 4152414 A

L52: Entry 10 of 10 File: USPT May 1, 1979

US-PAT-NO: 4152414
DOCUMENT-IDENTIFIER: US 4152414 A

TITLE: Combination vaccine for swine dysentery and method of use

DATE-ISSUED: May 1, 1979

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Harris; Delbert L.	Ames	IA		
Goodnow; Robert A.	Omaha	NE		
Glock; Robert D.	Ames	IA		
Kinyon; Joann M.	Ames	IA		

US-CL-CURRENT: 424/490; 424/203.1, 424/234.1, 424/262.1, 424/825

ABSTRACT:

A combination vaccine for increasing the resistance of swine to dysentery infection comprises killed cells of a virulent isolate of Treponema hyodysenteriae in combination with concentrated killed cells of Bacteroides vulgatus, or Fusobacterium necrophorum, or mixtures thereof. This combination vaccine can be adapted for either oral or parenteral administration. For parenteral administration preferably only Bacteroides vulgatus is used in combination with Treponema hyodysenteriae. The oral vaccine is enteric-coated.

10 Claims, 0 Drawing figures Exemplary Claim Number: 1

L52: Entry 10 of 10 File: USPT May 1, 1979

DOCUMENT-IDENTIFIER: US 4152414 A
TITLE: Combination vaccine for swine dysentery and method of use

ABPL:
A combination vaccine for increasing the resistance of swine to dysentery infection comprises killed cells of a virulent isolate of Treponema hyodysenteriae in combination with concentrated killed cells of Bacteroides vulgatus, or Fusobacterium necrophorum, or mixtures thereof. This combination vaccine can be adapted for either oral or parenteral administration. For parenteral administration preferably only Bacteroides vulgatus is used in combination with Treponema hyodysenteriae. The oral vaccine is enteric-coated.

BSPR:
For use as an oral vaccine, Fusobacterium necrophorum in the form of concentrated killed cells can be employed instead of or in addition to Bacteroides vulgatus. One preferred oral vaccine contains both Bacteroides vulgatus and Fusobacterium necrophorum cells with the T. hyodysenteriae antigen, and each are used in the relative proportions to T. hyodysenteriae specified above with respect to B. vulgatus.

BSPR:
For preparing vaccines in accordance with the present invention, it is believed that any strain or isolate of Bacteroides vulgatus or Fusobacterium necrophorum can be used. Strains of these bacteria which can be used in practicing the present invention are therefore readily available, such as those on deposit with the American Type Culture Collection, Rockville, Md. For B. vulgatus these include the type strain ATCC No. 8482, and other strains or isolates, such as ATCC No. 31376.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 1. Document ID: US 6309669 B1

L60: Entry 1 of 5 File: USPT Oct 30, 2001

US-PAT-NO: 6309669
DOCUMENT-IDENTIFIER: US 6309669 B1

TITLE: Therapeutic treatment and prevention of infections with a bioactive materials encapsulated within a biodegradable-biocompatible polymeric matrix

DATE-ISSUED: October 30, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Setterstrom; Jean A.	Alpharetta	GA		
Van Hamont; John E.	Fort Meade	MD		
Reid; Robert H.	McComas	CT		
Jacob; Elliot	Silver Spring	MD		
Jeyanthi; Ramasubbu	Columbia	MD		
Boedeker; Edgar C.	Chevy Chase	MD		
McQueen; Charles E.	Olney	MD		
Jarboe; Daniel L.	Silver Spring	MD		
Cassels; Frederick	Ellicott City	MD		
Brown; William	Denver	CO		
Thies; Curt	Ballwin	MO		
Tice; Thomas R.	Birmington	AL		
Roberts; F. Donald	Dover	MA		
Friden; Phil	Beford	MA		

US-CL-CURRENT: 424/486; 424/422, 424/423, 424/424, 424/425, 424/484

ABSTRACT:

Novel burst-free, sustained release biocompatible and biodegrable microcapsules which can be programmed to release their active core for variable durations ranging from 1-100 days in an aqueous physiological environment. The microcapsules are comprised of a core of polypeptide or other biologically active agent encapsulated in a matrix of poly(lactide/glycolide) copolymer, which may contain a pharmaceutically-acceptable adjuvant, as a blend of upcapped free carboxyl end group and end-capped forms ranging in ratios from 100/0 to 1/99.

25 Claims, 87 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 85

L60: Entry 1 of 5 File: USPT Oct 30, 2001

DOCUMENT-IDENTIFIER: US 6309669 B1
TITLE: Therapeutic treatment and prevention of infections with a bioactive materials encapsulated within a biodegradable-biocompatible polymeric matrix

BSPR:
Applicants contemplate that the invention will be useful in the formulation of disease specific compositions for the prevention and/or treatment of diseases and/or ailments which include: viral infections; bacterial infections; fungal infections; yeast infections; parastic infections and more specific diseases and/or ailments; such as as, aids; alzheimer's dementia; angiogenesis diseases; aphthour ulcers in AIDS patients; asthma; atopic dermatitis; psoriasis; basal cell carcinoma; benign prostatic hypertrophy; blood substitute; blood substitute in surgery patients; blood substitute in trauma patients; breast cancer; breast cancer; cutaneous & metastatic; cachexia in AIDS; campylobacter infection; Cancer; pnemonia; sexually transmitted diseases (STDs); cancer; viral dieases; candida albicians in AIDS and cancer; candidiasis in HIV infection; pain in cancer; pancreatic cancer; parkinson's disease; peritumoral brain edema; postoperative adhesions (prevent); proliferative diseases; prostate cancer; ragweed allergy; renal disease; restenosis; rheumatoid arthritis; rheumatoid arthritis; allergies; rotavirus infection; scalp psoriasis; septic shock; small-cell lung cancer; solid tumors; stroke; thrombosis; type I diabetes; type I diabetes w/kidney transplants; type II diabetes; viseral leishmaniasis; malaria; periodontal or gum disease; cardiac rthythm disorders; central nervous system diseases; central nervous system disorders; cervical dystonia (spasmodic torticollis); choridal neovascularization; chronic hepatitis c, b and a; colitis associated with antibiotics; colorectal cancer; coronary artery thrombosis; cryptosporidiosis in AIDS; cryptosporidium parvum diarrhea in AIDS; cystic fibrosis; cytomegalovirus disease; depression; social phobias; panic disorder; diabetic complications; disabetic eye disease; diarrhea associated with antibiotics; erectile dysfunction; genital herpes; graft-vs host disease in transplant patients; growth hormone deficiency; head and neck cancer; head trauma; stroke; heparin neutralization after cardiac bypass; hepatocellular carcinoma; HIV; HIV infection; huntington's disease; CNS diseases; hypercholesterolemia; hypertension; inflammation; inflammation and angiogensis; inflammation in cardiopulmonary bypass; influenza; migrain head ache; interstitial cystitis; kaposi's sarcoma; kaposi's sarcoma in AIDS; lung cancer; melanoma; molluscum contagiosum in AIDS; multiple sclerosis; neoplastic meningitis from solid tumors; non-small cell lung cancer; organ transplant rejection; osteoarthritis; rheumatoid arthritis; osteoporosis; drug addiction; shock; ovarian cancer; and pain.

DEPR:
Successful controlled release of bioactive ampicillin anhydrate was achieved in vitro and in vivo. The prototype microcapsules/spheres effectively controlled or eliminated Staphylococcus aureus and Steptococcus pyogenes from infected wounds in rats. Additionally, the formulation would be effective in the treatment of all bacterial infections caused by organisms sensitive to the antibiotic encapsulated including but not limited to Enterobacteriaceae; Klebsiella sp.; Bacteroides sp.; Enterococci; Proteus sp.; Streptococcus sp.; Staphylococcus sp.; Pseudomonas sp.; Neisseria sp.; Pedptostreptococcus sp.; Fusobacterium sp.; Actinomyces sp.; Mycobacterium sp.; Listeria sp.; Corynebacterium sp. ; Proprionibacterium sp.; Actinobacillus sp.; Aerobacter sp.; Borrelia sp.; Campylobacter sp.; Cytophaga sp.; Pasteurella sp.; Clostridium sp.; Enterobacter aeroqenes; Peptococcus sp.; Proteus vulgaris; Proteus morganii; Staphylococcus aureus; Streptococcus polygenes; Actinomyces sp.; Campylobacter fetus; and Legionella pneumophila. Results indicate that optimal microcapsules/spheres should exhibit a programmed release of an appropriate concentration of antibiotic over about a 14 day to about a 6 week time period after which time the microcapsule/sphere should biodegrade, leaving no trace of drug or excipient.

DEPV:
144. The method of Item 142 wherein the bacterial infection is caused by a resistant or non-resistant bacteria selected from the group consisting essentially of Enterobacteriaceae; Klebsiella sp.; Bacteroides sp. Enterococci; Proteus sp.; Streptococcus sp.; Staphylococcus sp.; Pseudomonas sp.; Neisseria sp.; Pedptostreptococcus sp.; Fusobacterium sp.; Actinomyces sp.; Mycobacterium sp.; Listeria sp.; Corynebacterium sp.; Proprionibacterium sp.; Actinobacillus sp.; Aerobacter sp.; Borrelia sp.; Campylobacter sp.; cytophaga sp.; Pasteurella

sp.; Clostridium sp., Enterobacter aerogenes, Peptococcus sp., Proteus vulgaris, Proteus morganii, Staphylococcus aureus, Streptococcus pyogenes, Actinomyces W., Campylobacter fetus, and Legionella pneumophila, ampillin-resistant strain of S. aureus, and methicillin-resistant strain of S. aureus.

DEPV:
155. The process of using the composition of Item 1 to treat humans in need, thereof, suffering from diseases and/or ailments from the group consisting of: viral infections; bacterial infections; fungal infections; parastic infections and more specific diseases and/or ailments; such as as, aids; alzheimer's dementia; angiogenesis diseases; aphthour ulcers in AIDS patients; asthma; atopic dermatitis; psoriasis; basal cell carcinoma; benign prostatic hypertrophy; blood substitute; blood substitute in surgery patients; blood substitute in trauma patients; breast cancer; breast cancer; cutaneous & metastatic; cachexia in AIDS; campylobacter infection; cancer; pnemonia; sexually transmitted diseases (STDs); cancer; viral dieases; candida albicians in AIDS and cancer; candidiasis in HIV infection; pain in cancer; pancreatic cancer; parkinson's disease; peritumoral brain edema; postoperative adhesions (prevent); proliferative diseases; prostate cancer; ragweed allergy; renal disease; restenosis; rheumatoid arthritis; rheumatoid arthritis; allergies; rotavirus infection; scalp psoriasis; septic shock; small-cell lung cancer; solid tumors; stroke; thrombosis; type I diabetes; type I diabetes w/kidney transplants; type II diabetes; viseral leishmaniasis; malaria; periodontal or gum disease; cardiac rthythm disorders; central nervous system diseases; central nervous system disorders; cervical dystonia (spasmodic torticollis); choridal neovascularization; chronic hepatitis c, b and a; colitis associated with antibiotics; colorectal cancer; coronary artery thrombosis; cryptosporidiosis in AIDS; cryptosporidium parvum diarrhea in AIDS; cystic fibrosis; cytomegalovirus disease; depression; social phobias; panic disorder; diabetic complications; disabetic eye disease; diarrhea associated with antibiotics; erectile dysfunction; genital herpes; graft-vs host disease in transplant patients; growth hormone deficiency; head and neck cancer; head trauma; stroke; heparin neutralization after cardiac bypass; hepatocellular carcinoma; HIV; HIV infection; huntington's disease; CNS diseases; hypercholesterolemia; hypertension; inflammation; inflammation and angiogensis; inflammation in cardiopulmonary bypass; influenza; migrain head ache; interstitial cystitis; kaposi's sarcoma; kaposi's sarcoma in AIDS; lung cancer; melanoma; molluscum contagiosum in AIDS; multiple sclerosis; neoplastic meningitis from solid tumors; non-small cell lung cancer; organ transplant rejection; osteoarthritis; rheumatoid arthritis; osteoporosis; drug addiction; shock; ovarian cancer; Amebiasis; Babesiasis; Chagas' disease Trypanosoma cruzi); Cryptosporidiosis; Cysticercosis; Fascioliasis; Filariasis; Echinococcosis; Giardiasis; Leishmaniasis; Malaria; Paragonimiasis; Pneumocystosis; Schistosomiasis; Strongylodiasis; Toxocariasis; Toxoplasmosis; Trichinellosis; Trichomoniasis; yeast infection; and pain.

DEPV:
156. A vaccine for prepared from the composition of Item 1 to prevent the occurence in humans of diseases and/or ailments comprising viral infections; bacterial infections; fungal infections; parastic infections and more specific diseases and/or ailments; such as as, aids; alzheimer's dementia; angiogenesis diseases; aphthour ulcers in AIDS patients; asthma; atopic dermatitis; psoriasis; basal cell carcinoma; benign prostatic hypertrophy; blood substitute; blood substitute in surgery patients; blood substitute in trauma patients; breast cancer; breast cancer; cutaneous & metastatic; cachexia in AIDS; campylobacter infection; cancer; pnemonia; sexually transmitted diseases (STDs); cancer; viral dieases; candida albicians in AIDS and cancer; candidiasis in HIV infection; pain in cancer; pancreatic cancer; parkinson's disease; peritumoral brain edema; postoperative adhesions (prevent); proliferative diseases; prostate cancer; ragweed allergy; renal disease; restenosis; rheumatoid arthritis; rheumatoid arthritis; allergies; rotavirus infection; scalp psoriasis; septic shock; small-cell lung cancer; solid tumors; stroke; thrombosis; type I diabetes; type I diabetes w/kidney transplants; type II diabetes; viseral leishmaniasis; malaria; periodontal or gum disease; cardiac rthythm disorders; central nervous system diseases; central nervous system disorders; cervical

dystonia (spasmodic torticollis); choridal neovascularization; chronic hepatitis c, b and a; colitis associated with antibiotics; colorectal cancer; coronary artery thrombosis; cryptosporidiosis in AIDS; cryptosporidium parvum diarrhea in AIDS; cystic fibrosis; cytomegalovirus disease; depression; social phobias; panic disorder; diabetic complications; disabetic eye disease; diarrhea associated with antibiotics; erectile dysfunction; genital herpes; graft-vs host disease in transplant patients; growth hormone deficiency; head and neck cancer; head trauma; stroke; heparin neutralization after cardiac bypass; hepatocellular carcinoma; HIV; HIV infection; huntington's disease; CNS diseases; hypercholesterolemia; hypertension; inflammation; inflammation and angiogensis; inflammation in cardiopulmonary bypass; influenza; migrain head ache; interstitial cystitis; kaposi's sarcoma; kaposi's sarcoma in AIDS; lung cancer; melanoma; molluscum contagiosum in AIDS; multiple sclerosis; neoplastic meningitis from solid tumors; non-small cell lung cancer; organ transplant rejection; osteoarthritis; rheumatoid arthritis; osteioporosis; drug addiction; shock; ovarian cancer; Amebiasis; Babesiasis; Chagas' disease (Trypanosoma cruzi); Cryptosporidiosis; Cysticercosis; Fascioliasis; Filariasis; Echinococcosis; Giardiasis; Leishmaniasis; Malaria; Paragonimiasis; Pneumocystosis; Schistosomiasis; Strongylodiasis; Toxocariasis; Toxoplasmosis; Trichinellosis; Trichomoniasis; yeast infection; and pain.

☐ 2. Document ID: US 5856302 A

L60: Entry 2 of 5 File: USPT Jan 5, 1999

US-PAT-NO: 5856302
DOCUMENT-IDENTIFIER: US 5856302 A

TITLE: Therapeutic uses of bactericidal/permeability-increasing protein dimer products

DATE-ISSUED: January 5, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ammons; William Steve	Pinole	CA		
Little; Roger G.	Benicia	CA		

US-CL-CURRENT: 514/12; 424/192.1, 514/2, 530/350

ABSTRACT:

Improved therapeutic compositions, methods and uses of bactericidal/permeability-increasing protein (BPI) products involve use of dimeric forms of BPI protein product monomers characterized by enhanced in vivo biological activity. Preferred formulations include 50 percent or more by weight dimeric product and preferred therapeutic uses include endotoxin neutralization and heparin neutralization.

16 Claims, 14 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 12

L60: Entry 2 of 5 File: USPT Jan 5, 1999

DOCUMENT-IDENTIFIER: US 5856302 A
TITLE: Therapeutic uses of bactericidal/permeability-increasing protein dimer products

BSPR:
Gram-negative bacteria include bacteria from the following species: Acidaminococcus, Acinetobacter, Aeromonas, Alcaligenes, Bacteroides, Bordetella, Branhamella, Brucella, Calymmatobacterium, Campylobacter, Cardiobacterium, Chromobacterium, Citrobacter, Edwardsiella, Enterobacter, Escherichia, Flavobacterium, Francisella, Fusobacterium, Haemophilus, Klebsiella, Legionella, Moraxella, Morganella, Neisseria, Pasturella, Plesiomonas, Proteus, Providencia, Pseudomonas, Salmonella, Serratia, Shigella, Streptobacillus, Veillonella, Vibrio, and Yersinia species.

BSPR:
The invention thus contemplates that administration of BPI protein product dimers will provide beneficial activity for neutralizing heparin, including neutralizing the anti-coagulant activity of heparin when administered in vivo to a subject. It is also contemplated that such products will be particularly useful when administered to subjects in order to inhibit endothelial cell proliferation and angiogenesis associated with a variety of conditions including malignant tumor proliferation, Kaposi's sarcoma lesions and the like. Cancers susceptible to treatment by administration of dimeric BPI protein products include melanoma, sarcomas, and carcinomas including, but not limited to, breast, colon, lung and prostate carcinomas. Other conditions for which dimeric BPI protein products can be administered for inhibition of angiogenesis include ocular retinopathy, retrolental fibroplasia, psoriasis, angiofibromas, endometriosis, hemangiomas and the like. Also contemplated by the invention are uses of dimeric BPI protein products in methods of contraception comprising delivering of an effective amount of a BPI protein product so as to prevent implantation of a fertilized ovum, methods of treating chronic inflammatory disease states such as arthritis, psoriasis, inflammatory bowel disease, Crohn's disease, ulcerative colitis, lupus erythematosus, autoimmune uveitis, Lyme disease, and asthma.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

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☐ 3. Document ID: US 5599795 A

L60: Entry 3 of 5 File: USPT Feb 4, 1997

US-PAT-NO: 5599795
DOCUMENT-IDENTIFIER: US 5599795 A

TITLE: Method for treatment of idiopathic inflammatory bowel disease (IIBD)

DATE-ISSUED: February 4, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McCann; Michael	Seven Hills	OH	44131	
Abrams; Richard S.	Wilmette	IL	60091	

US-CL-CURRENT: 424/93.4; 514/192, 514/198, 514/200, 514/210.04, 514/31, 514/398

ABSTRACT:

A new method for the prevention and treatment of Idiopathic inflammatory Bowel Disease (IIBD), including Crohn's Disease and Ulcerative Colitis, in human patients is provided. The key steps include sterilizing the intestinal tract with multiple antibiotics to kill the pre-existing bacterial flora, and replacing the latter with different, select, well-characterized bacteria taken from normal humans. The new microflora serve to generate more normal metabolic and immune responses, remitting thereby the IIBD.

3 Claims, 0 Drawing figures Exemplary Claim Number: 1

L60: Entry 3 of 5 File: USPT Feb 4, 1997

DOCUMENT-IDENTIFIER: US 5599795 A
TITLE: Method for treatment of idiopathic inflammatory bowel disease (IIBD)

ABPL:
A new method for the prevention and treatment of Idiopathic inflammatory Bowel Disease (IIBD), including Crohn's Disease and Ulcerative Colitis, in human patients is provided. The key steps include sterilizing the intestinal tract with multiple antibiotics to kill the pre-existing bacterial flora, and replacing the latter with different, select, well-characterized bacteria taken from normal humans. The new microflora serve to generate more normal metabolic and immune responses, remitting thereby the IIBD.

BSPR:
This invention relates to an improved method for preventing and treating idiopathic inflammatory bowel disease (IIBD), including Crohn's Disease and Ulcerative Colitis in human patients by sterilizing the intestinal tract by the use of multiple antibiotics, and the use of select, well-characterized intestinal bacteria taken from normal humans to replace the patient's intestinal bacteria.

DEPR:
Bacteria are obtained and maintained in freeze-dried (lyophilized) form, or frozen in liquid nitrogen, or in suitable culture media. The anaerobic bacteria require expert handling to avoid contamination from opportunistic bacteria to avoid damage from oxygen. Suitable sterile, commercially available oxygen-free solid or liquid culture media tubes or bottles are used to maintain the mixture of anaerobes. Aerobes are maintained with the same precision, in suitable sterile, solid or liquid, culture media tubes or bottles (for example, tryptocase soy broth) containing normal air. Materials: Specific strains of the following genera and species of bacteria will be used to accomplish the "re-florestation": Bacteroides vulgatus, distasonis, stercoris, ovatus, caccae, and uniformis; Bifidobactertum adolescentis, longum, and bifidum; Eubacterium aerofaciens, rectale; Fusobacterium prausnitzii; Ruminococcus obeum, bromii, gnavus; Lactobacillus acidophilus, leichmannii; Streptococcus oralis; Escherichia Coli (Pingel, Nissl 1917, and other strains).

CLPR:
1. A method of preventing or treating idiopathic inflammatory bowel disease, Crohn's Disease or ulcerative colitis in a human patient comprising administering to said patient a composition comprising at least one antibiotic agent and at least one anti-fungal agent to sterilize the intestine of said patient and then infusing orally and rectally to said patient a specific bacteria composition to establish colonization of the intestine whereby the pathological immune response is normalized and inflammation ceases.

CLPR:
3. The method of claim 1, wherein said bacteria composition consists essentially of Bacteroides caccae, distasonis, fragilis, stercoris, thetaiotaomicron, uniformis, vulgatus; Bifidobacterium adolescentis, longum coprococcus eutactus; Eubacterium aerofaciens, rectale; Fusobacterium prausnitzii; Lactobacillus acidophilus; Ruminococcus gnavus, bromii, obeum; Streptococcus intermedius, oralis, salivarius and Veillonella parvula.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

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☐ 4. Document ID: US 5443826 A

L60: Entry 4 of 5 File: USPT Aug 22, 1995

US-PAT-NO: 5443826
DOCUMENT-IDENTIFIER: US 5443826 A

TITLE: Treatment of gastro-intestinal disorders with a fecal composition or a composition of bacteroides and E. Coli

DATE-ISSUED: August 22, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Borody; Thomas J.	Five Dock, New South Wales		2046	AUX

US-CL-CURRENT: 424/93.3; 424/543, 424/93.48, 426/2, 426/61, 426/71

ABSTRACT:

A method of treating chronic disorders associated with the presence of abnormal microflora or an abnormal distribution of microflora in the gastrointestinal tract. The method involves the removal of at least a portion of the host's existing enteric microflora and the substitution of an effective amount of predetermined microflora. Pharmaceutical compositions which comprise viable microorganisms in a composition resembling the host's normal healthy faecal flora are also disclosed.

6 Claims, 0 Drawing figures Exemplary Claim Number: 1

L60: Entry 4 of 5 File: USPT Aug 22, 1995

DOCUMENT-IDENTIFIER: US 5443826 A
TITLE: Treatment of gastro-intestinal disorders with a fecal composition or a composition of bacteroides and E. Coli

BSPR:
There are large numbers of patients suffering from gastro-intestinal symptoms referrable to the lower small bowel and large bowel which to date have eluded explanation. These disorders include irritable bowel syndrome (IBS) or spastic

colon, idiopathic ulcerative colitis, mucous colitis, collagenous colitis, Crohn's disease, inflammatory bowel disease in general, microscopic colitis, antibiotic-associated colitis, idiopathic or simple constipation, diverticular disease, and AIDS enteropathy. Pathophysiology of these disorders eludes logical explanation in spite of decades of research and millions of dollars of research funds. A common underlying factor shared by all these disorders observed by the present inventor is their onset following some extraneous invading infection. In all the disorders, the infection cannot be demonstrated due to our inability to detect infecting agents whose cultural characteristics are unknown to medical science.

BSPR:
The present invention recognises chronic infection/infestation as the underlying pathological process in a wide range of chronic disorders such as irritable bowel syndrome, particularly when characterised by chronic abdominal pain, bloating, or excessive flatulence, together with chronic diarrhoea or alternating constipation/diarrhoea, and also in spastic colon, mucous colitis, collagenous colitis, ulcerative colitis, Crohn's colitis, microscopic colitis, idiopathic inflammatory bowel disease, antibiotic-associated colitis, idiopathic or simple constipation, diverticular disease and AIDS enteropathy.

BSPR:
In a preferred form the synthetic faecal composition comprises a preparation of viable flora which preferably in proportional content, resembles normal healthy human faecal flora. Suitable microorganisms may be selected from the following; Bacteroides, Bifidobacterium, Eubacteria, Fusobacteria, Propionibacteria, Lactobacilli, anaerobic cocci, Ruminococcus, E. Coli, Gemmiger, Clostridium, Desulfomonas, species and, more specifically, bacteria selected from Table 1. Preferably fungi are also present such as Monilia.

BSPR:
In practice suitable microorganisms include those selected from Bacteroides, Bifidobacterium, Eubacteria, Fusobacteria, Propionibacteria, Lactobacilli, anaerobic cocci, Ruminococcus, E. coli, Gemmiger, Clostridium, Desulfomonas, and Monilia species, and more specifically from those set out in Table 1.

BSPR:
As indicated in the method of treatment aspect of the invention, a preparatory course of appropriate antibiotics may be used. For example, Septrin for chronic yersiniasis, Metronidazole for ulcerative colitis, anti-TB therapy in Crohn's disease, or Vancomycin in chronic Clostridium difficile infestations.

BSPV:
i) gastro-intestinal disorders including irritable bowel syndrome or spastic colon, ulcerative colitis, mucous colitis, collagenous colitis, Crohn's disease, inflammatory bowel disease, microscopic colitis, antibiotic associated colitis, idiopathic or simple constipation, diverticular disease, AIDS enteropathy, small bowel bacterial overgrowth, coeliac disease, polyposis coli, colonic polyps, chronic idiopathic pseudo obstructive syndrome;

BSTL:
TABLE 1 _____ % of florab Organism(s)
_____ 11.8(0.90) Bacteroides fragilis ss. vulgatus 9.9(0.83) Eubacterium aerofaciens 8.9(0.78) Bacteroides fragilis ss. thetaiotaomicron 6.6(0.68) Peptostreptococcus productus II 6.0(0.64) Bacteroides fragilis ss. distasonis 4.4(0.55) Fusobacterium prausnitzii 3.5(0.49) Coprococcus eutactus 3.0(0.45) Eubacterium aerofaciens III 2.8(0.44) Peptostreptococcus productus I 2.7(0.43) Ruminococcus bromii 2.6(0.43) Bifidobacterium adolescentis 2.2(0.39) Gemmiger formicilis, Bifidobacterium longum 2.1(0.38) Eubacterium siraeum 1.8(0.35) Ruminococcus torques 1.7(0.34) Eubacterium rectale III-H 1.6(0.33) Eubacterium rectale IV, Eubacterium eligens 1.5(0.32) Bacteroides eggerthii 1.4(0.31) Clostridium leptum 1.3(0.29) Bacteroides fragilis ss. a 1.2(0.29) Eubacterium biforme 0.91(0.25) Bifidobacterium infantis 0.84(0.24) Eubacterium rectale III-F 0.57(0.20) Coprococcus comes, Bacteroides capillosus 0.50(0.18) Ruminococcus albus,

Eubacterium formicigenerans, Eubacterium hallii, Eubacterium ventriosum I, Fusobacterium russii 0.43(0.17) Ruminococcus obeum, Eubacterium rectale II, Clostridium ramosum I, Lactobacillus leichmanii 0.36(0.16) Ruminococcus callidus, Butyrivibrio crossotus 0.30(0.14) Acidaminococcus fermentans, Eubacterium ventriosum, Bacteroides fragilis ss. fragilis, Bacteroides AR 0.23(0.12) Coprococcus catus, Eubacterium hadrum, E. cylindroides, E. ruminantium, Eubacterium CH-1, Staphylococcus epidermidis 0.17(0.10) Peptostreptococcus BL, Eubacterium limosum, Bacteroides praeacutus, Bacteroides L, Fusobacterium mortiferum I, F. naviforme, Clostridium innocuum, C. ramosum, Propionibacterium acnes, Ruminococcus flavefaciens 0.10(0.08) Ruminococcus AT, Peptococcus AU-1, Eubacterium AG, -AK, -AL, -AL-1, -AN; Bacteroides fragilis ss. ovatus, -ss. d, -ss. f; Bacteroides L-1, L-5; Fusobacterium nucleatum, F. mortiferum, Esherichia coli, Streptococcus morbillorum 0.05(0.05) Peptococcus magnus, Peptococcus G, -AU-2; Strepto- coccus intermedius, Ruminococcus lactaris, Ruminococcus CO, Gemmiger X, Coprococcus BH, CC; Eubacterium tenue, Eubacterium ramulus, Eubacterium AE, -AG-H, -AG-M, -AJ, -BW-1; Bacteroides clostridiiformis ss. clostridiiformis, B. coagulans, B. oralis, B. ruminicola ss. brevis, -ss. ruminicola, Bacteroides splanchnicus, Desulfomonas pigra, Bacteroides L-4, -W-1; Fusobacterium H, Lactobacillus G, Succinivibrio A

DEPR:
Similar methods were used to treat 55 patients suffering with either constipation, diarrhoea, abdominal pain, ulcerative colitis or Crohn's disease. Patients were treated when other forms of therapy had failed to control their symptoms. Following bowel flora alteration 20 patients were deemed "cured", 9 improved, while 26 failed to improve. The following cases illustrate "cures" using the method of the invention over a follow up period of 1 to 12 months.

DEPR:
This 45 year old man presented with an 18 month history of ulcerative colitis and elevated liver transaminases. Pancolitis was confirmed on colonoscopy. Sulfasalazine caused a rash while olsalazine gave inadequate relief. The patient underwent exchange of bowel flora improving adequately enough to come off treatment within days. At three months he continues to feel well, has no diarrhoea and is on no medication. His liver transaminases have returned to normal. Clonoscopy is now normal and mucosal biopsies are now normal.

DEPL:
Case 4 Chronic Ulcerative Colitis

CLPR:
1. A method for the treatment of chronic gastrointestinal disorder selected from the group consisting of ulcerative colitis, Crohn's disease, and irritable bowel syndrome, in a human host, which method comprises the removal of at least the portion of the host's existing enteric microflora that is removed using a lavage and the substitution of an effective amount of fresh or dried or reconstituted feces from a disease-screened human donor or a composition comprising Bacteroides and Escherichia coli species in liquid culture or dried viable form.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 5407682 A

L60: Entry 5 of 5 File: USPT Apr 18, 1995

US-PAT-NO: 5407682
DOCUMENT-IDENTIFIER: US 5407682 A

TITLE: Process for the preparation of azo-and /or disulfide polymer matrix drug delivery system for the site specific delivery of an active agent in the colon

DATE-ISSUED: April 18, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Schacht; Etienne	Staden			BEX
Wilding; Ian	Bramcote			GBX

US-CL-CURRENT: 424/436; 424/426, 424/468, 424/482, 424/486, 424/497

ABSTRACT:

Azo-and/or disulfide-containing polymers for use as drug delivery systems having site-specific release of the drug in the colon are obtained by polycondensation or polyaddition of an azo- and/or polysulfide disulfide containing a, w-dihydroxy or diamino reagent with a suitable a, w-difunctional dicarboxylic acid; disocyanato-, disulfide comonomer. The resulting reduction sensitive polymers are linear macromolecules containing an azo and/or a disulfide bond in their polymer backbone.

12 Claims, 2 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 1

L60: Entry 5 of 5 File: USPT Apr 18, 1995

DOCUMENT-IDENTIFIER: US 5407682 A
TITLE: Process for the preparation of azo-and /or disulfide polymer matrix drug delivery system for the site specific delivery of an active agent in the colon

BSPR:
Peppercorn et al. published in Journal of Pharmacology and Experimental Therapy, 181, 555, 1972 that salicylazosulfapyridine (a drug applied for the therapeutic treatment of ulcerative colitis) (azuflidine, sulfasalazine) can be cleaved by microflora in the colon with release of 5-aminosalicylic acid.

BSPR:
The disulfide bond in glutathione drug conjugates can be reduced in the colon and lead to liberation of the glutathion-linked thiol (G. L. Larsen, J. P. Larson, J. A. Gustarson, Fusobacterium necrophorum, Xenobiotica, 13, 689, 1983).

Full | Title | Citation | Front | Review | Classification | Date | Reference
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JPAB,EPAB,DWPI	antigen	35182	L6
JPAB,EPAB,DWPI	l1 and l6	13	L7
JPAB,EPAB,DWPI	l7 not (oral or periodontal).ti.	7	L8
JPAB,EPAB,DWPI	antibody	62901	L9
JPAB,EPAB,DWPI	l1 and l9	13	L10

JPAB,EPAB,DWPI	110 not 17	9	L11
JPAB,EPAB,DWPI	111 not (oral or periodontal).ti.	7	L12
JPAB,EPAB,DWPI	adhesin or adhesion	197895	L13
JPAB,EPAB,DWPI	11 and 113	1	L14
JPAB,EPAB,DWPI	intestin3n with mucos3n	0	L15
JPAB,EPAB,DWPI	intestinal with mucosa	317	L16
JPAB,EPAB,DWPI	11 and 116	0	L17
JPAB,EPAB,DWPI	vaccin$	21053	L18
JPAB,EPAB,DWPI	11 and 118	17	L19
JPAB,EPAB,DWPI	119 not (17 or 110 or 114)	9	L20
JPAB,EPAB,DWPI	butyric or butyrate	8523	L21
JPAB,EPAB,DWPI	11 and 121	0	L22
JPAB,EPAB,DWPI	11 and 13	0	L23
JPAB,EPAB,DWPI	13 and 121	30	L24
JPAB,EPAB,DWPI	13 with 121	8	L25
JPAB,EPAB,DWPI	(animal or disease) with model	2449	L26
JPAB,EPAB,DWPI	126 and 11	0	L27
JPAB,EPAB,DWPI	126 and 13	39	L28
JPAB,EPAB,DWPI	13 same 126	21	L29
JPAB,EPAB,DWPI	13 with 126	4	L30
JPAB,EPAB,DWPI	129 not (17 or 110 or 114 or 119 or 125)	21	L31
JPAB,EPAB,DWPI	129 and 121	0	L32
JPAB,EPAB,DWPI	128 and 121	0	L33
JPAB,EPAB,DWPI	L3 with ulcer$	3017	L34
JPAB,EPAB,DWPI	126 same 134	20	L35
JPAB,EPAB,DWPI	diagnos$	130558	L36
JPAB,EPAB,DWPI	136 and 11	3	L37
JPAB,EPAB,DWPI	137 not (17 or 110 or 114 or 119 or 125)	1	L38
JPAB,EPAB,DWPI	137 and 134	0	L39
JPAB,EPAB,DWPI	136 and 134	382	L40
JPAB,EPAB,DWPI	135 and 136	14	L41
JPAB,EPAB,DWPI	126 with 134	4	L42
JPAB,EPAB,DWPI	142 not (17 or 110 or 114 or 119 or 125 or 137)	4	L43

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Search Results - Record(s) 1 through 6 of 6 returned.

☐ 1. Document ID: JP 2001046063 A

L2: Entry 1 of 6 File: JPAB Feb 20, 2001

PUB-NO: JP02001046063A
DOCUMENT-IDENTIFIER: JP 2001046063 A
TITLE: PRIMER FOR GENUS EUBACTERIUM AND FUSOBACTERIUM VARIUM BACTERIUM

PUBN-DATE: February 20, 2001

INVENTOR-INFORMATION:

NAME	COUNTRY
BENNO, YOSHIMI	
KAGEYAMA, AKIKO	

INT-CL (IPC): C12N 15/09; C12Q 1/68

ABSTRACT:

PROBLEM TO BE SOLVED: To obtain the subject primer for identifying a bacterium belonging to the genus Eubacterium rapidly, simply at a low cost in a high accuracy, useful for preventing/treating various diseases without culturing a bacterium by making the primer have a specific base sequence or a sequence complementary to the base sequence.

SOLUTION: This primer has a base sequence selected from formula I to formula IV or a base sequence complementary to the base sequence. A primer or a probe for a Fusobacterium varium group bacterium, having a base sequence base of taccggatat tatgactgag or a base sequence complementary to the base sequence is prepared. A primer having a combination of a base sequence of formula I or formula II and a base sequence of formula III or formula IV, etc., or a combination of base sequences complementary to these sequences, for detecting the species of bacterium belonging to the genus Eubacterium is preferably prepared. The species of bacterium belonging to the genus Eubacterium or a Fusobacterium varium group bacterium is preferably identified by using these primers.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Desc | Clip Img | Image

☐ 2. Document ID: JP 04330016 A

L2: Entry 2 of 6 File: JPAB Nov 18, 1992

PUB-NO: JP404330016A
DOCUMENT-IDENTIFIER: JP 04330016 A
TITLE: LIPOPEROXIDE HYPOLIPIDEMIC AGENT

PUBN-DATE: November 18, 1992

INVENTOR-INFORMATION:

NAME	COUNTRY
SUZUKI, KUNIO	

INT-CL (IPC): A61K 35/66; A23L 1/03; A61K 35/68; A61K 35/70; A61K 35/72; A61K 35/74; A61K 35/74; A61K 35/74; A61K 35/76 ; A61K 35/80; C12N 1/20

ABSTRACT:

PURPOSE: To provide a low-toxic lipoperoxide reducer for degrading the lipoperoxides taken in or produced in the body to effect detoxication taking advantage of microorganisms capable of degrading lipoperoxides such as mammal's intestinal microorganisms.

CONSTITUTION: The objective lipoperoxide hypolipidemic agent containing, as active ingredient, microorganisms capable of decomposing lipoperoxides, in particular, mammal's intestinal microorganisms belonging to Bacteroides, Fusobacterium, Escherichia, Clostridium, Eubacterium or Beijerinckia, among others, Bacteroides vulgatus S-602, Fusobacterium varium VII-16, Fusobacterium varium VII-59, Escherichia coli O-601, Escherichia coli F-604, Clostridium bifermentans B-1, or Eubacterium tenu X-10. The present medicine is useful for the prevention of such diseases as arteriosclerosis, hepatitis, hepatocirrhosis, jecur adiposum, pulmonary emphysema, pulmonary fibrosis, digestive tract ulcer, myocardial infarction, cerebral apoplexy, cataract, amyloidosis and chronic arthrorheumatism.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Desc | Image

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☐ 3. Document ID: RU 2170510 C2

L2: Entry 3 of 6 File: DWPI Jul 20, 2001

DERWENT-ACC-NO: 2001-474672
DERWENT-WEEK: 200151

TITLE: Method of preparing biological preparation ekofit for protection of plants against phytopathogens and for harvest increase

INVENTOR: ASHCHEULOV, V I; EGOROVA, N G ; GOLUBEV, O A ; KUZNETSOV O YU, ; MARKOV, V S ; RUPASOV, K I

PRIORITY-DATA: 1999RU-0114412 (July 1, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
RU 2170510 C2	July 20, 2001		000	A01N063/00

INT-CL (IPC): A01N 63/00; C12P 39/00

ABSTRACTED-PUB-NO: RU 2170510C
BASIC-ABSTRACT:

NOVELTY - Preparation for plant protection against phytopathogens and for harvest increase is prepared by separate culturing bacterial strains of species Pseudomonas fluorescens, Eubacterium tortuosum, Alcaligenes xylosoxidans, Clostridium innocuum, Prevotella intermedia, Eubacterium saburreum, Pseudomonas aeruginosa, Pseudomonas mendocina, Fusobacterium varium, Clostridium sp., Bacillus subtilis. Obtained cultures are mixed, inoculated in liquid nutrient medium and cultured at stirring up to titer value 500 million cells/ml, not less, and mother culture is obtained. The latter is inoculated in industrial nutrient medium and cultured. Invention can be used in production of preparations showing phytoprotective effect, correction of soil microflora and improvement its structure.

USE - Biotechnology, agriculture.

ADVANTAGE - Protection of plants and harvest against phytopathogens, stimulation of seed germination, improved plant nutrition.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Desc | Image

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☐ 4. Document ID: JP 2001046063 A

L2: Entry 4 of 6 File: DWPI Feb 20, 2001

DERWENT-ACC-NO: 2001-294439
DERWENT-WEEK: 200131

TITLE: Primers for Eubacterium sp. and Fusobacterium varium group.

PRIORITY-DATA: 1999JP-0226176 (August 10, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2001046063 A	February 20, 2001		009	C12N015/09

INT-CL (IPC): C12N 15/09; C12Q 1/68

ABSTRACTED-PUB-NO: JP2001046063A
BASIC-ABSTRACT:

NOVELTY - Primers or probes (I) useful for rapid and simple detection of Eubacterium sp. and Fusobacterium varium group microorganisms and having any of the 19 fully defined 20-22 nucleotide sequences given in the specification or their complements, are new.

DETAILED DESCRIPTION - Primers or probes (I) for Eubacterium sp. microorganisms having any of the 18 fully defined 20-22 nucleotide sequences given in the specification or their complements, particularly combinations of (S1) or (S2) and (S11) or (S12); (S3) and (S13); (S4) and (S14); (S5) and (S15); (S6) and (S16); (S7) and (S17); (S8) and (S18) or (S9) and (S19), or their complements

or for F. varium group microorganisms having the fully defined 20 nucleotide sequence (S10) given in the specification, or its complement

USE - (I) are useful for detection and identification of Eubacterium sp. and F. varium group microorganisms in the intestine or oral cavity of animals including humans.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 5. Document ID: JP 03197483 A

L2: Entry 5 of 6 File: DWPI Aug 28, 1991

DERWENT-ACC-NO: 1991-298768
DERWENT-WEEK: 199141

TITLE: New penem b-sulphoxide derivatives - are antimicrobials against gram-positive and -negative bacterial e.g. S. aureus and E. coli and anaerobes

PRIORITY-DATA: 1989JP-0339077 (December 27, 1989)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 03197483 A	August 28, 1991		000	

INT-CL (IPC): C07D 499/88

ABSTRACTED-PUB-NO: JP03197483A
BASIC-ABSTRACT:

Derivs. of formula (I) or their salts are new. In (I), R1=H, aryl, or heterocycle which contains at least one O or N atom; R2=H, alkyl, aryl, aralkyl, or allyl; R3= alkyl or opt. protected hydroxyalkyl; and n=0-10.

(I) may be administered orally or parenterally at a daily dose of 50 mg - 5g., pref. 100 mg - 4 g., for an adult.

USE - (I) show potent antimicrobial action against gram-positive bacteria (e.g. Strapylococcus aureus, Micrococcus luteus), gram-negative bacteria (e.g. Escherichia coli, Klebsiella pneumoniae, Serratia marcescens, Proteus morganii, Enterobactor cloacae, Alcaligenes faecalis, Proteus vulgaris), and anaerobes (e.g. Bacteroides fragilis, Fusobacterium varium) at a conc. of 0.025-50 mcg/ml. (I) have dehydropeptidase-resistant activity.

(I) may be prepd. by oxidn. of penem derivs. of formula (II) with a peracid e.g. AcOOH, monochlorophthalic acid, m-chloroperbenzoic acid (MCP), metaperiodic acid, in an organic solvent e.g. AcOH, CH2Cl2, at a temp. lower than 10 deg. C.

In an example, to a soln. of 88g (0.2mmol) allyl (5R,6S)-6-((R)-1-(tert,- butyl di-methysilyloxy)ethyl)-2 -((R)-tetrahydro-2-furanyl) penem-3-carbox ylate in 0.5 ml CH2Cl2 was added a soln. of 43mg(0.2 mmol) MCP in 0.5ml CH2Cl2 with ice cooling. The mixt. was stirred at the same temp. for 20 mins. then diluted with CH2Cl2, washed with 5% Na2S2O3 and H2O, dried on MgSO4, and evaporated. The residue was applied to silica gel chromatography to give 48.3 mg (53% yield) allyl (5R,6S)-6-((R)-1-tert-butyl dimethylsilyloxy)ethyl)-2-((R)-tetrahydro-2-furanyl) penem sulphoxide-3-carboxylate as colourless oil.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 6. Document ID: EP 277688 A, US 5578470 A, NL 8700240 A, JP 63240793 A, US 5182194 A, EP 277688 B1, DE 3885115 G, ES 2061622 T3

L2: Entry 6 of 6 File: DWPI Aug 10, 1988

DERWENT-ACC-NO: 1988-221513
DERWENT-WEEK: 199702

TITLE: Prepn. of thiol cpds. - by conjugating cpd. with cysteine and reacting the prod. conjugate with beta-lyase

INVENTOR: BERG, J; KERKENAAR, A ; SCHMEDDING, D J M

PRIORITY-DATA: 1987NL-0000240 (January 30, 1987)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 277688 A	August 10, 1988	E	014	
US 5578470 A	November 26, 1996		016	C12P011/00
NL 8700240 A	August 16, 1988		000	
JP 63240793 A	October 6, 1988		000	
US 5182194 A	January 26, 1993		010	C12P011/00
EP 277688 B1	October 27, 1993	E	013	C12P011/00
DE 3885115 G	December 2, 1993		000	C12P011/00
ES 2061622 T3	December 16, 1994		000	C12P011/00

INT-CL (IPC): A23L 1/22; A23L 1/226; C07B 45/06; C07C 45/78; C07C 145/00; C07C 319/02; C07J 31/00; C12N 9/88; C12P 7/26 ; C12P 11/00; C12P 13/12; C12P 33/00

ABSTRACTED-PUB-NO: EP 277688A
BASIC-ABSTRACT:

A method for prepg. thiol cpds. is characterised in that cysteine is coupled via an -S-bridge to a hydrocarbon cpd. and subsequently the cysteine conjugate obtd. is reacted with a beta-lyase to form one or more thiol cpds.

Pref. the cysteine is coupled by addn. to an alpha, beta-unsatd. aldehyde or ketone of formula R1R2C=C(R3)-CO-R4 (R1-R4=H or an opt. satd. and/or heterogeneous hydrocarbon gp. or together with the C atoms to which they are bonded form 1 or 2, opt. satd. and/or heterogeneous ring systems) and subsequently the conjugate obtd. is reacted with beta-lyase to form the thiol cpds.

Also claimed is a flavour compsn. comprising P-mentha-8-thiol -3-one, together with customary ingredients. Also claimed is a flavour compsn. comprising a flavouring cpd. of formula (I) (R5 = H, 1-24C alkyl or an alkaline ion; R6 = a gp. consisting of 1-7 monosaccharides selected from glucose, mannose, galactose, arabinose, fucose, xylose, rhamnose, uronic acids as well as the acetates, pyruvates, amines and sulphates derived from these) together with customary ingredients.

Also claimed is a method for purifying or sepg. aldehydes and ketones, in partic. alpha, beta-unsatd. aldehydes and ketones, from prods. contg. such carbonyl cpds., characterised in that cysteine is bonded to the respective aldehyde or ketone via an S-bridge and the cysteine conjugate obtd. is isolated and then split into cysteine and the respective aldehyde or ketone.

USE/ADVANTAGE - The purification can be carried out under mild conditions and the cysteine is recovered. Many types of thiol cpds. can be prepd. e.g., perfumes and flavouring (P-mentha-8-thiol-3-one, damascone deriv.) pharmacological steroid cpds. (e.g., 16-mercaptoprogesterone) and repellants (Warburganal).
ABSTRACTED-PUB-NO:

EP 277688B EQUIVALENT-ABSTRACTS:

Method for preparing thiol compounds, characterized in that cysteine is coupled by means of addition to a compound having the formula (R1)(R2)C = C(R3)-Co-R4 in which the symbols R1-R4 represent a hydrogen or an optionally saturated and/or heterogeneous hydrocarbon group or, together with the carbon atoms to which the symbols are bonded, form one or two, optionally saturated and/or heterogeneous hydrocarbon ring systems and that subsequently the cysteine conjugate obtained is reacted with beta-lyase to form the relevant thiol compounds.

US 5182194A

Prepn. of thiol cpds. comprises reacting cysteine non-enzymatically with pulegone to form a cysteine conjugate cpd. This is then reacted with a beta-lyase chosen from Eubacterium limosum (pref.), Fusobacterium necrophorum and Fusobacterium varium beta-lyases to produce p-mentha-8-thi ol-3-one (I), which is recovered. USE - (I) is a perfume. The method may also be used in the prodn. of flavourings, pharmaceutical steroids and repellants.

US 5578470A

A method for preparing thiol compounds, comprising:

(1) reacting cysteine by a non-enzymatic addition reaction with a compound having the formula (R1)(R2)C=C(R3)-CO-R4 via an -S- bridge to form a cysteine conjugate,

wherein R1, R2 and R3 are each selected from the group consisting of: hydrogen; an alkyl group containing 1-5 carbon atoms; an alkylene group containing 2-6 carbon atoms; a cycloalkyl or cycloalkenyl group containing 5-10 carbon atoms and an aryl group containing 6-10 carbon atoms; and R4 is selected from the group consisting of: hydrogen; an alkyl group containing 1-5 carbon atoms; an alkylene group containing 2-6 carbon atoms; a cycloalkyl or cycloalkenyl group containing 5-10 carbon atoms; an aryl group containing 6-10 carbon atoms; -OH and -OCH3, or

wherein a combination of two groups selected from the group consisting of R1, R3 and R4, together with the carbon atoms to which the groups are bonded, form a ring system of five or six members, wherein said ring has 0-3 ethenic unsaturated bonds and wherein said ring has 0-2 heterogeneous atoms selected from the group consisting of N and O, or

wherein said compound having the formula (R1)(R2)C=C(R3)-CO-R4 is a compound having the formula:

in which the R5 is selected from the group consisting of hydrogen, an alkyl containing 1-24 carbon atoms and an alkaline ion, and R6 is 1-7 monosaccharides and wherein said monosaccharides are selected from the group consisting of glucose, mannose, galactose, arabinose, fucose, xylose, rhamnose, uronic acid, and acetate, pyruvate, amine and sulphate derivatives thereof, and

(2) reacting said cysteine conjugate in a concentration of greater than 1 mM conjugate with a cysteine conjugate beta-lyase produced by bacteria selected from the group consisting of Eubacterium limosum, Escherichia coli, Fusobacterium varium, Fusobacterium nucleatum, Salmonella typhimurium, Enterobacter cloacae, Bacillus brevis, Pseudomonas taetrolens, Pseudomonas aromatica and Pseudomonas fluorescens to form a thiol compound and recovering said thiol compound.

Full | Title | Citation | Front | Review | Classification | Date | Reference

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Term	Documents
FUSOBACTERIUM.DWPI,EPAB,JPAB.	168
FUSOBACTERIUMS	0
FUSOBACTERIA.DWPI,EPAB,JPAB.	5
FUSOBACTERIAS	0
VARIUM.DWPI,EPAB,JPAB.	23
VARIUMS	0
VARIA.DWPI,EPAB,JPAB.	182
VARIAS.DWPI,EPAB,JPAB.	3
F.DWPI,EPAB,JPAB.	2212159
FS.DWPI,EPAB,JPAB.	5673
(FUSOBACTERIUM VARIUM OR F VARIUM).JPAB,EPAB,DWPI.	6

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☐ 1. Document ID: AU 732317 B, WO 9805755 A1, AU 9738099 A, EP 863981 A1, CN 1198185 A, JP 2000500027 W, NZ 330050 A, BR 9706543 A, MX 9802543 A1, KR 99063786 A, US 6162429 A

L8: Entry 1 of 7 File: DWPI Apr 12, 2001

DERWENT-ACC-NO: 1998-145598
DERWENT-WEEK: 200128

TITLE: New bacterium Serpens sp. HBL-112, associated with papillomatous digital dermatitis in ruminants - and derived vaccines and diagnostic assays for detecting specific antigens or antibodies

INVENTOR: WALLIS, D; WALLIS, J L ; WILLIS, D

PRIORITY-DATA: 1996US-0022915 (August 1, 1996), 1997US-0903559 (July 31, 1997)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 732317 B	April 12, 2001		000	C12N001/00
WO 9805755 A1	February 12, 1998	E	052	C12N001/00
AU 9738099 A	February 25, 1998		000	C12N001/00
EP 863981 A1	September 16, 1998	E	000	C12N001/00
CN 1198185 A	November 4, 1998		000	C12N001/00
JP 2000500027 W	January 11, 2000		041	C12N001/20
NZ 330050 A	January 28, 2000		000	C12N001/04
BR 9706543 A	December 28, 1999		000	C12N001/00
MX 9802543 A1	November 1, 1998		000	C12N001/00
KR 99063786 A	July 26, 1999		000	C12N001/20
US 6162429 A	December 19, 2000		000	A01N063/00

INT-CL (IPC): A01N 63/00; A01N 65/00; A61K 5/00; A61K 35/74; A61K 39/02; A61K 51/00; A61P 31/00; C12N 1/00; C12N 1/04; C12N 1/12; C12N 1/20; C12N 1/36; C12R 1/01; G01N 33/53; G01N 33/554; G01N 33/569

ABSTRACTED-PUB-NO: US 6162429A
BASIC-ABSTRACT:

Biologically pure Serpens spp. strain HBL-112 (A) is new. Also claimed are: (1) a composition for preventing or treating papillomatous digital dermatitis (PDD) in ruminants containing a Serpens spp. bacteria or Serpens spp. bacterin and/or their immunologically active fragments and/or an epitope cross-reactive with Serpens spp., plus a carrier or diluent; and (2) a method for detecting PDD-associated antigen or antibody in ruminant serum or anti-Serpens antibody in ruminants.

Compositions of (1) contain (A) or S. flexibilis (live or dead), or their bacterins. Vaccines may be made multivalent, e.g. they also include Bacteroides and/or Fusobacterium antigens. PDD antibodies are detected by treating a sample

with an antigen, e.g. Serpens bacterium or bacterin, active fragment or cross-reactive epitope, then detecting complex formation. Anti-Serpens antibodies are detected by adding anti-ruminant antibody, labelled with enzyme, and suitable enzyme substrate. Serpens antigen are detected by complex formation with anti-Serpens antibody. Complex formation is detected from a label in the primary binding partner or by reaction with second, labelled binding partner.

USE - The compositions of (1) induce a protective immune response against PDD in cattle, sheep and goats. The methods of (2) are used to diagnose PDD. Also (not claimed) Serpens antigen can be used to purify Ab and vice versa. Compositions are administered subcutaneously, parenterally or orally. Typical doses are 108-1011 (particularly 109-1010), plaque-forming units/ml, in a 5 ml subcutaneous injection, with 2 or 3 doses given at 3-4 week intervals.
ABSTRACTED-PUB-NO:

WO 9805755A EQUIVALENT-ABSTRACTS:

Biologically pure Serpens spp. strain HBL-112 (A) is new. Also claimed are: (1) a composition for preventing or treating papillomatous digital dermatitis (PDD) in ruminants containing a Serpens spp. bacteria or Serpens spp. bacterin and/or their immunologically active fragments and/or an epitope cross-reactive with Serpens spp., plus a carrier or diluent; and (2) a method for detecting PDD-associated antigen or antibody in ruminant serum or anti-Serpens antibody in ruminants.

Compositions of (1) contain (A) or S. flexibilis (live or dead), or their bacterins. Vaccines may be made multivalent, e.g. they also include Bacteroides and/or Fusobacterium antigens. PDD antibodies are detected by treating a sample with an antigen, e.g. Serpens bacterium or bacterin, active fragment or cross-reactive epitope, then detecting complex formation. Anti-Serpens antibodies are detected by adding anti-ruminant antibody, labelled with enzyme, and suitable enzyme substrate. Serpens antigen are detected by complex formation with anti-Serpens antibody. Complex formation is detected from a label in the primary binding partner or by reaction with second, labelled binding partner.

USE - The compositions of (1) induce a protective immune response against PDD in cattle, sheep and goats. The methods of (2) are used to diagnose PDD. Also (not claimed) Serpens antigen can be used to purify Ab and vice versa. Compositions are administered subcutaneously, parenterally or orally. Typical doses are 108-1011 (particularly 109-1010), plaque-forming units/ml, in a 5 ml subcutaneous injection, with 2 or 3 doses given at 3-4 week intervals.

Full | Title | Citation | Front | Review | Classification | Date | Reference

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☐ 2. Document ID: RU 2098128 C1

L8: Entry 2 of 7 File: DWPI Dec 10, 1997

DERWENT-ACC-NO: 1998-360873
DERWENT-WEEK: 199831

TITLE: Vaccine against limb infection disease in sheep - contains specified bacteria cultures and adjuvants

INVENTOR: KIRILLOV, L V; KRUZHNOV, N N ; PANASYUK, S D

PRIORITY-DATA: 1996RU-0119161 (September 26, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
RU 2098128 C1	December 10, 1997		006	A61K039/116

INT-CL (IPC): A61K 39/116; C12N 1/20; A61K 39/116; A61K 39/02; A61K 39/05; A61K 39/08; A61K 39/085; C12N 1/20; C12R 1/04 ; C12R 1/145; C12R 1/15; C12R 1/445

ABSTRACTED-PUB-NO: RU 2098128C
BASIC-ABSTRACT:

A vaccine comprises inactivated cultures (including antigens) of Bacteroides nodosus, Fusobacterium necrophorum, Staphylococcus aureus and Clostridium perfringens. The vaccine additionally comprises salt solution, adjuvant and additionally contains anatoxins of: (a) Clostridium perfringens type A VGNKI 28-DEP; (b) inactivated culture of Actinomyces (Coryne-bacterium) pyogenes VGNKI N4/2334-DEP, (c) B. nodosus VIEV N PP82/90, (d)F. necrophorum BGNKI N Neva-DEP (e) Fusobacterium necrophorum VGNKI N Kr-1-DEP, (f) Staphylococcus aureus VGNKI n 7315-DEP, and/or (g) Clostridium perfringens type A VGNKI N 28-DEP.

The cultures are inactivated with formalin and the adjuvant is in form of aluminium hydroxide gel.

USE - The vaccine can be used in biotechnology and veterinary microbiology . The vaccine is named Ovikon and is for preventing and treating limb infection disease of sheep.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 3. Document ID: RU 2098127 C1

L8: Entry 3 of 7 File: DWPI Dec 10, 1997

DERWENT-ACC-NO: 1998-360872
DERWENT-WEEK: 199831

TITLE: Associated vaccine against necrobacteriosis of cattle - contains complex of bacteria cultures inactivated with formalin, and additionally physiological solution

INVENTOR: PANASYUK, S D; SIDORUK, A A ; USTINOVA, G I

PRIORITY-DATA: 1996RU-0119157 (September 26, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
RU 2098127 C1	December 10, 1997		006	A61K039/116

INT-CL (IPC): A61K 39/116; C12N 1/20; A61K 39/116; A61K 39/02; A61K 39/05; A61K 39/08; A61K 39/085; C12N 1/20; C12R 1/04 ; C12R 1/145; C12R 1/15; C12R 1/445

ABSTRACTED-PUB-NO: RU 2098127C
BASIC-ABSTRACT:

A vaccine comprises bacteria inactivated with formalin. The bacteria are in the form of cultures of Fusobacterium necrophorum, Staphylococcus aureus, Corynebacterium pyogenes, Clostridium perfringens type A. The vaccine additionally comprises an adjuvant, salt solution, and antigens from: Fusobacterium necrophorum VGNKI N Neva-DEP (I), Fusobacteri um necrophorum VGNKI N Kr-1-DEP (II), Staphylococcus aureus VGNKI N 7315-DEP (III), Actinomyces (Corynebacterium) pyogenes VGNKI N 4/2334-DEP (IV), Clostridium perfringens type A VGNKI N 28-DEP (V), and/or anatoxin. The adjuvant is in the form of a gel of aluminium hydroxide, and additionally contains immuno-stimulant GMDP.

USE - The vaccine can be used in biotechnology and veterinary microbiology . The new vaccine (commercial nameNekovak) can be used for for active immunisation of cattle against limb necrobacteriosis disease.

ADVANTAGE - The vaccine has improved activity and stability, and increased immunogenic range.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 4. Document ID: EP 764446 A2

L8: Entry 4 of 7 File: DWPI Mar 26, 1997

DERWENT-ACC-NO: 1997-181645
DERWENT-WEEK: 199717

TITLE: Serum-based vaccine contg. adjuvant and no non-host albumin - to prevent adverse reactions to non-host albumin

INVENTOR: BROWN, K K; HENNESSY, K J ; LANE, J K ; TRUMP, S L

PRIORITY-DATA: 1996EP-0114505 (September 11, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 764446 A2	March 26, 1997	E	017	A61K039/23

INT-CL (IPC): A61K 39/02; A61K 39/12; A61K 39/23

ABSTRACTED-PUB-NO: EP 764446A

BASIC-ABSTRACT:

A serum-based vaccine comprising an immunogenically effective amt. of an antigen and an adjuvant, where the vaccine is substantially free of non-host albumin (i.e. contains less than 1 mg/ml of non-host albumin), is new. Also claimed are: (1) a method for preparing a vaccine as above, comprising removing non-host albumin from the vaccine or a precursor of the vaccine; (2) a vaccine as above also contg. host albumin; (3) a method for preparing the vaccine of (2) by: (a) growing an organism which produces the antigen in a culture contg. host albumin; (b) harvesting the culture; (c) clarifying the harvest if necessary; and (d) formulating the harvest with an adjuvant; (4) a serum based-vaccine comprising an immunogenically effective amt. of an antigen, an adjuvant and a host serum or host albumin which is added after harvest or purificn. of the antigen but prior to adjuvanting the antigen; (5) a method for preparing a serum-based vaccine contg. an immunogenically effective amt. of an antigen and an adjuvant by adding a host serum or host albumin after harvesting or purifying the antigen from a culture contg. the antigen but prior to adjuvanting the antigen; (6) a process for stabilising an antigen comprising adding host serum or host albumin to the antigen prior to adjuvanting the antigen; (7) a process for stabilising an antigen comprising adding host serum or host albumin to the antigen prior to lyophilising the antigen; and (7) a method for purificn. of veterinary vaccine antigens comprising perfusion chromatography.

The antigen is a virus, bacterium, rickettsia, parasite or protozoon or their subunits. The virus is selected from retroviruses, herpes viruses, adenoviruses, paramyxoviruses, coronaviruses, morbilliviruses, hantaviruses, reoviruses, rotaviruses, togaviruses, parvoviruses, parapox viruses, cytomegaloviruses, arboviruses and parainfluenza viruses. The bacterium is selected from Clostridium, Streptococcus, Staphylococcus, Bordetella, Pasteurella, Salmonella, Mycobacterium, Mycoplasma, Leptospira, Borrelia, Fusobacterium, Bacteroides, Rhodococcus, Escherichia, Moraxella and Haemophilus spp. The rickettsia is a Chlamydia or Ehrlichia sp. The parasite is a Toxoplasma, Dirofilaria, Cryptosporidium, Coccidia, Babesia or Neospora sp. The adjuvant is selected from polymers, block copolymers, oils, o/w emulsions, Al salts and non-specific immunostimulants. The adjuvant is present in an amt. of 0.01-50%.

ADVANTAGE - Adverse vaccine reactions induced by non-host albumin are reduced or prevented.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

☐ 5. Document ID: RU 2043770 C1

L8: Entry 5 of 7 File: DWPI Sep 20, 1995

DERWENT-ACC-NO: 1996-220174
DERWENT-WEEK: 199622

TITLE: Obtaining vaccine against necrobacteriosis in animals - using Fusobacteriu m necrophorum cultures and rupturing the cells with proteolytic enzyme, e.g. proto-subtilin

INVENTOR: LAVCHENKO, E G; SOLOMAKHA, O I ; ZHIROV, V A

PRIORITY-DATA: 1993RU-0008462 (February 15, 1993)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
RU 2043770 C1	September 20, 1995		004	A61K039/02

INT-CL (IPC): A61K 39/02

ABSTRACTED-PUB-NO: RU 2043770C
BASIC-ABSTRACT:

Obtaining a vaccine against necrobacteriosis in animals comprises the separate growing of Fusobacterium necrophorum (FN) cultures (serotypes I and II), sepn. of the biomass, rupture of the microbe cells, sepn. of the complex water soluble antigens from the liq. fraction, standardisation of the antigens obtd., mixing of these in different protein ratios according to the quantity of protein, addn. of an adjuvant-adsorbent (AA) and prodn. of the desired prod., is new. The microbe cells are ruptured by means of a proteolytic enzyme, the sepd. antigens are inactivated with formaldehyde and a 3% soln. of aluminium hydroxide is used as the AA. The desired prod. contains (vol. %): complex of water soluble FN antigen serotype I (30-40), complex of water soluble FN antigen serotype II (30-40), formalin (0.3-0.4) and 3% Al hydroxide soln. (25-30).

USE - The method gives a highly effective inactivated vaccine against necrobacteriosis in animals.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw. Desc | Image

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☐ 6. Document ID: RU 2040273 C1

L8: Entry 6 of 7 File: DWPI Jul 27, 1995

DERWENT-ACC-NO: 1996-149585
DERWENT-WEEK: 199615

TITLE: Cattle necrobacillosis antigen prepn. - by growing virulent Fusobacterium necrophorum, inactivating, freeze-thawing, homogenising and centrifuging

INVENTOR: ALEKSEEVA, I I; KAMALOV G KH, KHUZIN|S A ; ,

PRIORITY-DATA: 1993RU-0025043 (May 28, 1993)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
RU 2040273 C1	July 27, 1995		004	A61K039/02

INT-CL (IPC): A61K 39/02; C12N 1/20

ABSTRACTED-PUB-NO: RU 2040273C
BASIC-ABSTRACT:

A cattle necrobacillosis antigen of high specificity and immunogenicity can be synthesised by using locally-occurring strains of the pathogen which have the optimum absorbence and virulence w.r.t. laboratory animals. A Fusobacterium necrophorum strain that causes death 24-48 hrs. after intraperitoneal injection in mice is cultivated until (1-2). 107 microbial cells/cm3 have appeared. The antigen is extracted by means of a 3-stage freezing-thawing lysis, then homogenised in aq. medium. After centrifugation at 15000-20000 g, the end prod. is isolated as the cytoplasmic fraction from the supernatant.

USE - The method is used in veterinary microbiology, veterinary science, specifically to obtain cattle necrobacillosis antigen.

ADVANTAGE - The antigen produced provides immunity in 50-80% of white mice 14-20 days after inoculation and protects cattle from necrobacillosis infection for up to 6 months.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 7. Document ID: SU 1835295 A1

L8: Entry 7 of 7 File: DWPI Aug 23, 1993

DERWENT-ACC-NO: 1995-073838
DERWENT-WEEK: 199510

TITLE: A vaccine for prevention of necrobacteriosis in cattle - contg. formalin-inactivated Staphylococcus aureus and Clostridium perfringens type A antigens

INVENTOR: KOROMYSLOV, G F; SIPORCHUK, A A ; USTANOVA, G T

PRIORITY-DATA: 1990SU-4864625 (June 22, 1990)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
SU 1835295 A1	August 23, 1993		004	A61K039/02

INT-CL (IPC): A61K 37/02; A61K 39/02

ABSTRACTED-PUB-NO: SU 1835295A
BASIC-ABSTRACT:

A vaccine for prevention of necrobacteriosis in cattle, including formalin-inactivated Fusobacterium necrophorum (IFN) and Corynebacterium pyogenes (ICP) antigens, is new. The vaccine contains the formalin-inactiv ated antigens Staphylococcus aureus and Clostridium perfringens type A anatoxin (CPAA), aluminium hydroxide, and N-acetylglycosaminyl (beta 1-4)-N-acetylmuramyl-alanine-D-isoglutamine (I), 100% soln. in the following proportions (ml): 40.0-50.0 IFN contg. 1-2.109cells per ml, 20.0-30.0 ICP contg. 109 cells per ml, 40.0-50.0 formalin-inactiva ted S. aureus antigen contg. 2.109cells per ml, 60.0-70.0 formalin-inactiv ated CPAA contg. 2.109 cells per ml, 5.0-6.0 aluminium hydroxide, 60-240 (I), 100% soln. of CPAA (activity 15-20 units in 1 m) with 0.2% formalin to 1000.

USE - The vaccine together with (I) activates the phagocytosis system, which is a central linkage in the development of immunological reactions.

ADVANTAGE - The method gives a vaccine giving increased intensity of immunity to necrobacterium stimulants, and having a wider spectrum of prophylactic action.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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Term	Documents
ORAL.DWPI,EPAB,JPAB.	12366
ORALS	0
PERIODONTAL.DWPI,EPAB,JPAB.	1057
PERIODONTALS	0
(7 NOT ((PERIODONTAL OR ORAL).TI.)).JPAB,EPAB,DWPI.	7

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☐ 1. Document ID: JP 03127996 A

L12: Entry 1 of 7 File: JPAB May 31, 1991

PUB-NO: JP403127996A
DOCUMENT-IDENTIFIER: JP 03127996 A
TITLE: MONOCLONAL ANTIBODY AGAINST CARCINOSTATIC SUBSTANCE TF-300

PUBN-DATE: May 31, 1991

INVENTOR-INFORMATION:

NAME	COUNTRY
MURAKAMI, SHOHACHI	
SUGITA, MASATOSHI	
MIYAURA, TATSUYA	
YONEZAWA, MINORU	

US-CL-CURRENT: 435/948
INT-CL (IPC): C12P 21/08; G01N 33/577; C12N 5/20; C12N 15/06

ABSTRACT:

PURPOSE: To provide a monoclonal antibody against a salt of a carcinostatic substance TF-300 produced by separating a carcinostatic fraction from a cultured liquid of a microbial strain belonging to genus Fusobacterium and treating the fraction with a proteinase and useful for the immunoassay of the salt for the detection and quantitative determination.

CONSTITUTION: The objective monoclonal antibody exhibits specific affinity to the above carcinostatic substance and its salt. The antibody is light gray or pale brown powder, inhibits the proliferation of Ehrlich's ascites carcinoma cell, Ehrlich's nodular carcinoma, sarcoma 180 cell and B-16 melanoma cell and has immuno-activating activity. The antibody can be prepared by conventional immunization process, fusion process, screening process and cloning process.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

☐ 2. Document ID: US 6291178 B1

L12: Entry 2 of 7 File: DWPI Sep 18, 2001

DERWENT-ACC-NO: 2001-647225
DERWENT-WEEK: 200174

TITLE: Kit for collecting and preserving saliva samples for analysis comprises a solution containing an ionic solute and an enzyme inhibitor

INVENTOR: SCHNEIDER, D R

PRIORITY-DATA: 1999US-0385171 (August 30, 1999), 1997US-0978729 (November 26, 1997)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 6291178 B1	September 18, 2001		009	C12Q001/68

INT-CL (IPC): C12Q 1/68

ABSTRACTED-PUB-NO: US 6291178B
BASIC-ABSTRACT:

NOVELTY - An assay sample collection kit for collection and preservation of a saliva sample for the assay of a biological analyte comprising an ionic solute which yields a solution with the osmolality of a normal physiological body fluid when diluted with water, and an inhibitor of a saliva enzyme that utilizes the analyte as a substrate, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) detecting a biological analyte in plasma, comprising:

(i) collecting a saliva sample at a first location directly into a specimen cup containing a measured aqueous solution of an enzyme inhibitor in pH-buffered saline;

(ii) sealing the cup against spillage and tampering;

(iii) transporting the cup to a second location; and

(iv) assaying the sample and solution for the analyte at the second location; and

(2) assaying for a biological analyte, comprising sampling a saliva preservative solution to detect an analyte selected from virus-infected cells, bacteria, carbohydrates, nucleic acids, lipids, fatty acids, melanin, hormones, steroids, cholesterol, insulin, glucogen, antibodies, polypeptides, blood groups, proteins, neurotransmitters, prostaglandin and other metabolites.

USE - The kit is useful for collecting and preserving saliva samples for subsequent assay for virus-infected cells (especially infected with human immunodeficiency virus (HIV), hepatitis, herpes or influenza viruses, rhinoviruses, adenoviruses, enteroviruses or picornaviruses), bacteria (especially tuberculosis bacilli, pneumococci, Klebsiella, Streptococcus, Staphylococcus, Mycobacterium, Bordetella, Corynebacterium, Clostridium, Fusobacterium, Escherichia, spirochaetes, Salmonella, Enterobacterium, Shigella or Brucella), carbohydrates, nucleic acids, lipids, fatty acids, melanin, hormones (especially growth hormone releasing factor, endocrine, hypothalamic, pituitary or adrenal hormones, sex hormones or gastrointestinal hormones), steroids, cholesterol, insulin, glucogen, antibodies, polypeptides, blood groups, proteins, neurotransmitters, prostaglandin and other metabolites.

ADVANTAGE - The enzyme inhibitor inhibits degradation of the analyte between sample collection and analysis.

☐ 3. Document ID: EP 1104308 A1, WO 200009164 A1, NL 1009834 C2, AU 9953882 A

L12: Entry 3 of 7 File: DWPI Jun 6, 2001

DERWENT-ACC-NO: 2000-224186
DERWENT-WEEK: 200133

TITLE: Antibody which binds to an epitope, where the binding can be broken under certain pH conditions useful for targeted and temporary diagnostic, therapeutic and cosmetic treatment of externally body parts

INVENTOR: HORBACH, D A; RENGGLI, H H ; WOUTERS, S L J

PRIORITY-DATA: 1998NL-1009834 (August 10, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1104308 A1	June 6, 2001	E	000	A61K047/48
WO 200009164 A1	February 24, 2000	E	018	A61K047/48
NL 1009834 C2	February 11, 2000		000	A61K047/48
AU 9953882 A	March 6, 2000		000	A61K047/48

INT-CL (IPC): A61K 7/16; A61K 7/28; A61K 47/48; C07K 16/00; C12P 21/00; G01N 33/563

ABSTRACTED-PUB-NO: WO 200009164A
BASIC-ABSTRACT:

NOVELTY - An antibody (I) or its fragments, which binds to an epitope under specifically chosen conditions, and the binding of which is broken under specifically chosen different conditions, is new.

ACTIVITY - Anti-carie; antibacterial.

MECHANISM OF ACTION - None given.

USE - (I) is used for targeted and temporary diagnostic, therapeutic and cosmetic treatment of externally accessible parts of the human and animal body. An externally accessible part of the human or animal body is the oral cavity and the cosmetic treatment of the oral cavity comprises the bleaching of teeth and molars. The diagnostic treatment of the oral cavity comprises the detection of plaque. The therapeutic treatment of the oral cavity comprises the removal of plaque. Alternatively, the therapeutic treatment comprises the treatment of infections in externally accessible parts of the human or animal body. (I) may be targeted against epitopes of pathogenic microorganisms (e.g. Actinomyces actinomycetem comitans, Porphyromonas gingivalis, Prevotella intermedia, Streptococcus mutans, Bacteroides forsythus, Eikenella corrodens, Treponema denticola, Campylobacter lectus or Fusobacterium nucleatum) or other pathogenic compounds (all claimed).

ADVANTAGE - Prior art methods for detecting dental plaque have involved the use of dyes conjugated to an antibody. However, the antibody-dye binding is not easily broken and dyes are visible to the eye for a longer time than with the use of (I). In addition, treatment of bacterial infections using (I) is more specific than using systemic antibiotics which spread throughout the entire body and kill

natural microbial gut flora of e.g. the gastrintestinal tract.

☐ 4. Document ID: KR 99022534 A, WO 9640237 A1, AU 9662709 A, EP 837695 A1, US 5861162 A, JP 11507377 W, BR 9610775 A, AU 713257 B, MX 9709785 A1

L12: Entry 4 of 7 File: DWPI Mar 25, 1999

DERWENT-ACC-NO: 1997-065145
DERWENT-WEEK: 200023

TITLE: Inoculum for preventing liver abscesses in ruminants contg. inactivated Actinomyces pyogenes - and opt. inactivated Fusobacterium necrophorum for additional control of foot rot, in cattle and sheep

INVENTOR: CHENGAPPA, M M; NAGARAJA, T G

PRIORITY-DATA: 1995US-0483382 (June 7, 1995), 1995US-0477619 (June 7, 1995)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
KR 99022534 A	March 25, 1999		000	A61K039/02
WO 9640237 A1	December 19, 1996	E	022	A61K039/02
AU 9662709 A	December 30, 1996		000	A61K039/02
EP 837695 A1	April 29, 1998	E	000	A61K039/02
US 5861162 A	January 19, 1999		000	A61K039/116
JP 11507377 W	June 29, 1999		025	A61K039/05
BR 9610775 A	July 13, 1999		000	A61K039/02
AU 713257 B	November 25, 1999		000	A61K039/02
MX 9709785 A1	October 1, 1998		000	A61K039/02

INT-CL (IPC): A61K 31/195; A61K 31/65; A61K 35/74; A61K 39/02; A61K 39/05; A61K 39/116; C12N 1/00; C12N 1/02; C12N 1/14

ABSTRACTED-PUB-NO: US 5861162A
BASIC-ABSTRACT:

Inoculum (A) for admin. to cattle or sheep to reduce incidence of liver abscesses comprises an inactivated cell culture prod. of Actinomyces pyogenes plus a carrier.

Also claimed is a similar inoculum (B) to reduce incidence of liver abscesses and/or foot rot that additionally contains an inactivated cell culture prod. of Fusobacterium necrophorum.

USE - The inocula (vaccines) are partic. administered to animals at high risk of developing abscesses and/or foot rot, i.e. those that regularly receive antibiotics or, in the case of cattle, are fed an all-concentrate diet contg. at least 95 wt.% grain. A. pyogenes and F. necrophorum are the two bacteria most commonly isolated from liver abscesses and foot rot lesions (A. pyogenes also causes pyogenic infections in other organs) and in combination they may be more virulent than either species alone. Vaccines are administered conventionally, pref. parenterally, one or more times to elicit the prodn. of protective antibodies.

ABSTRACTED-PUB-NO:

WO 9640237A EQUIVALENT-ABSTRACTS:

Inoculum (A) for admin. to cattle or sheep to reduce incidence of liver abscesses comprises an inactivated cell culture prod. of Actinomyces pyogenes plus a carrier.

Also claimed is a similar inoculum (B) to reduce incidence of liver abscesses and/or foot rot that additionally contains an inactivated cell culture prod. of Fusobacterium necrophorum.

USE - The inocula (vaccines) are partic. administered to animals at high risk of developing abscesses and/or foot rot, i.e. those that regularly receive antibiotics or, in the case of cattle, are fed an all-concentrate diet contg. at least 95 wt.% grain. A. pyogenes and F. necrophorum are the two bacteria most commonly isolated from liver abscesses and foot rot lesions (A. pyogenes also causes pyogenic infections in other organs) and in combination they may be more virulent than either species alone. Vaccines are administered conventionally, pref. parenterally, one or more times to elicit the prodn. of protective antibodies.

☐ 5. Document ID: US 5055405 A

L12: Entry 5 of 7 File: DWPI Oct 8, 1991

DERWENT-ACC-NO: 1991-317650
DERWENT-WEEK: 199143

TITLE: New MAB JD-1/18 produced by hybridoma cell line JD-1/18 - used to assay Treponema denticola JD-1 and Treponema species 10A

INVENTOR: BIEMER, T A; MINK, R ; STURDIVANT, L D ; WANG, D M

PRIORITY-DATA: 1987US-0123015 (November 19, 1987)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 5055405 A	October 8, 1991		000	

INT-CL (IPC): C07K 15/28; C12N 5/20; C12N 15/02; C12P 21/08

ABSTRACTED-PUB-NO: US 5055405A
BASIC-ABSTRACT:

Hybridoma cell line JD-1/18 is claimed, which produces an IgG3 antibody specific for Treponema denticola JD-1 (A) and Treponema species 10A (B). The cell line is formed by fusing a non-secreting mouse myeloma cell with a spleen cell from a mouse immunised with lysed cells of (A). Also claimed is the MAb JD-1/18 specific for the above microorganisms. The MAb is produced by fusing a non-secreting mouse myeloma cell Sp2.0-Ag14 with a spleen cell from a Balb/c mouse immunised with lysed cells of (A). The MAb is claimed to have no cross-reactivity with 28 listed microorganisms. Examples include T. denticola ATCC 33520, T. pectinovorum P5, T. vincentii N9, Actinomyces viscosus, Bacteriodes asaccharolyticus, Fusobacterium nucleatum, Streptococcus pyogenes, T. socranskii (subspecies buccale D-2B-8) and T. scoliodontum.

USE/ADVANTAGE - The MAb is used in immunoassays to detect the presence of (A) and

(B) in the oral cavity and to assay oral diseases in which (A) and (B) are implicated.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw. Desc | Image

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☐ 6. Document ID: JP 03127996 A

L12: Entry 6 of 7 File: DWPI May 31, 1991

DERWENT-ACC-NO: 1991-203815
DERWENT-WEEK: 199128

TITLE: Monoclonal antibody for anticancer substance TF-300 - obtd. by culturing Fusobacterium bacteria, useful for detection and assay

PRIORITY-DATA: 1989JP-0263755 (October 9, 1989)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 03127996 A	May 31, 1991		000	

INT-CL (IPC): C12N 5/20; C12N 15/06; C12P 21/08; C12R 1/91; G01N 33/57

ABSTRACTED-PUB-NO: JP03127996A
BASIC-ABSTRACT:

Monoclonal antibody shows specific affinity against anticancer substance TF-300 and its salts TF-300 is obtd by culturing Fusobacterium sp bacteria, then enzyme treatment of anticancer effect with fraction, obtd from cultured soln with protease, and has properties of a) white-grey or light brown powder, b) it inhibits growth of mouse Ehrlich's ascites- and Ehrlich's nodular-carcinomas sarcoma 180 and B-16 melanoma-cells, and has immuno potentiating effect, c) soluble in H20, insol in MeOH, EtOH acetone, benzene, CHCl3, EtOAc and diethyl ether, d) it does not show exact mpt, decomposed ca 180 deg C - 195 deg C-, e) IR(KBr Tab); 3500-3300, 2920, 2850, 1660-1620, 1580-1540, 1460-1400, 1380-1360, 1120, 1080-1020, 970, 820-800 (per cm), f) UV (H20; pH 7), strong absorption at absorption terminal, and near 246-280 nm, g) positive against Molisch, phenol--sulphonic- and anthrone sulphuric-acids, indol-HCl and Lowry-Folin, negative against Ninhydrin, h) elemental analysis; C of 38-47%, H of 5-7% and N of 1-4% i) content of saccharide by phenol sulphonic acid method is 16-60% (calculated, as glucose), content of protein by Lowry-Floin method is below calculated 10% (as bovine serum albumin).

USE/ADVANTAGE - Used for detection and assay of anticancer substance TF-300 and its salts. (7pp dwg.No.0/0)

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw. Desc | Image

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☐ 7. Document ID: JP 01313438 A, JP 95116059 B2

L12: Entry 7 of 7 File: DWPI Dec 18, 1989

DERWENT-ACC-NO: 1990-034387
DERWENT-WEEK: 199603

TITLE: ANti-parodontopathy compsn. - comprises chicken egg antibody to bond specifically to pathogenic bacteria of parodontopathy

PRIORITY-DATA: 1988JP-0145489 (June 13, 1988)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 01313438 A	December 18, 1989		004	
JP 95116059 B2	December 13, 1995		003	A61K039/395

INT-CL (IPC): A61K 7/16; A61K 39/39; A61K 39/395

ABSTRACTED-PUB-NO: JP01313438A
BASIC-ABSTRACT:

The compsn. contains chicken egg antibody to bond specifically to pathogenic bacteria of parodontopathy.

USE/ADVANTAGE - Effective for prevention of parodontopathy; partic. the compsn. inhibits fixing of bacteria to cause parodontopathy such as Bacteroides singinalis into oral cavity and propagation of the bacteria to prevent parodentopathyl. As pathogenic bacteris of parodontopathy may be cited Bacteroides singinalis, Actinobacillus actinomycetemcomitans, Fusobacterium nucieatum and Bacteroides melaninoger icus subsp. melaninogenicus etc.

In an example Bacteroides gingivalis GAI 7802 was cultured in GAM bouillon culture medium (Nissue Seiyaku Co.'s prod.), resultant bacteria was inactivated with 0.5% formalin in the usual manner to prepare vaccine.

Full | Title | Citation | Front | Review | Classification | Date | Reference

KWIC | Draw Desc | Image

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Term	Documents
ORAL.DWPI,EPAB,JPAB.	12366
ORALS	0
PERIODONTAL.DWPI,EPAB,JPAB.	1057
PERIODONTALS	0
(11 NOT ((PERIODONTAL OR ORAL).TI.)).JPAB,EPAB,DWPI.	7

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☐ 1. Document ID: JP 3042713 B2, WO 9211015 A1, EP 563256 A1, JP 06511469 W, US 5411948 A, EP 563256 B1, DE 69110906 E

L14: Entry 1 of 1 File: DWPI May 22, 2000

DERWENT-ACC-NO: 1992-249839
DERWENT-WEEK: 200029

TITLE: Use of host cell phospholipid(s) to bind microorganisms adhesion molecules - for treating and preventing microbial infections by preventing colonisation of epithelial cells

INVENTOR: KRIVAN, H C; LINGWOOD, C A ; NILSSON, B

PRIORITY-DATA: 1990US-0632372 (December 21, 1990), 1993US-0078474 (June 16, 1993)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 3042713 B2	May 22, 2000		012	A61K031/685
WO 9211015 A1	July 9, 1992	E	028	A61K031/685
EP 563256 A1	October 6, 1993	E	000	A61K031/685
JP 06511469 W	December 22, 1994		009	C07F009/10
US 5411948 A	May 2, 1995		008	A61K031/685
EP 563256 B1	June 28, 1995	E	019	A61K031/685
DE 69110906 E	August 3, 1995		000	A61K031/685

INT-CL (IPC): A61K 31/685; A61K 31/70; A61K 31/715; A61K 37/20; A61K 37/22; A61K 38/00; A61P 31/04; C07F 9/10; C07H 15/10; C12N 1/00; A61K 31/70; A61K 31/685; A61K 31/70; A61K 31/685; A61K 31/70; A61K 31/685

ABSTRACTED-PUB-NO: EP 563256B
BASIC-ABSTRACT:

Inhibiting microbial colonisation in a biological prepn. comprises contacting the biological prepn. with a phospholipid of formula (I) (where X = -CO-R or =CH=CH-R1; y = -CO-R; R1 = alkyl; R = an alkyl, hydroxyalkyl or alkenyl gp. of a fatty acid).

(I) were isolated from the lipid fraction of Hela cells and shown to bind with Chlamydial organisms.

USE/ADVANTAGE - The phospholipids (I) are receptors for microorganisms and bind microorganisms specifically and with high avidity.They can be used for treating and preventing microbial infections by preventing colonisation in vivo or in vitro.
ABSTRACTED-PUB-NO:

US 5411948A EQUIVALENT-ABSTRACTS:

A method for inhibiting bacterial colonization in a biological preparation,

comprising: contacting said biological preparation suspected

of containing bacteria selected from the group consisting of Streptococcus, Chlamydia, Clostridium, Borrella, Haemophilus, Pseudomonas, Neisseria, Helicobacter, Shigella, Pasteurella, Coxiella, Mycobacterium, Salmonella, Fusobacterium, Bacteroides and Campylobacter, with an effective amount of phospholipid having the formula (I) wherein X is -CO-R or -CH=CH-R'; Y is -COR; and R' is an alkyl group and R is an alkyl, hydroxyalkyl or alkenyl group of fatty acids.

Method comrpises contacting a biological prepn. suspected of contg. bacteria with an effective amt. of phospholipid receptor of formula (I) for sufficient time to allow bacteria to bind specifically to and with (I), thus preventing binding of bacteria to a native receptor on a host cell.

Suspect bacteria comprise Streptococcus, chlamydia, clostridium, Staphylococcus, Borrelia, Haemophilus, Pseudomonas, Neisseria, Helicobacter, Shigella, Pasteurella, Coxiella, Mycobacterium, Salmonella, Fusobacterium, Bacteroides or Compylobacter.

In (I), X is -C(O)R or -CH=CH-R'; Y is -C(O)R; R' is alkyl; and each R is (hydroxy)alkyl or alkenyl of fatty acid.

USE/ADVANTAGE - In vivo or in vitro inhibition of microbial colonisation. Versatile. Improves hygiene.

WO 9211015A

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Search Results - Record(s) 1 through 9 of 9 returned.

☐ 1. Document ID: US 5492694 A

L20: Entry 1 of 9 File: EPAB Feb 20, 1996

PUB-NO: US005492694A
DOCUMENT-IDENTIFIER: US 5492694 A
TITLE: Fusobacterium leukotoxoid vaccine

PUBN-DATE: February 20, 1996

INVENTOR-INFORMATION:

NAME	COUNTRY
NAGARAJA, TIRUVOOR G	US
CHENGAPPA, MUCKATIRA M	US

INT-CL (IPC): A61K 39/00; A61K 39/02
EUR-CL (EPC): C07K014/195

ABSTRACT:

A method is provided for the enhanced elaboration of leukotoxin from F. necrophorum, and subsequent production of an inactivated leukotoxoid ruminant animal vaccine against F. necrophorum infection and consequent liver abscesses and/or foot rot in such animals. The method involves forming a culture of F. necrophorum bacteria in growth media, allowing the bacteria to grow therein and to simultaneously elaborate leukotoxin in a supernate; the culturing is preferably carried out at a temperature of from about 35 DEG -41 DEG C., a pH of from about 6.5-8, and for a period of from about 4-9 hours. At the end of the culturing, bacterial growth and leukotoxin elaboration are terminated, preferably by separating the leukotoxin supernate, whereupon the vaccine is produced by inactivation of at least the supernate.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

☐ 2. Document ID: US 5455034 A

L20: Entry 2 of 9 File: EPAB Oct 3, 1995

PUB-NO: US005455034A
DOCUMENT-IDENTIFIER: US 5455034 A
TITLE: Fusobacterium necrophorum leukotoxoid vaccine

PUBN-DATE: October 3, 1995

INVENTOR-INFORMATION:

NAME	COUNTRY
NAGARAJA, TIRUVOOR G	US
CHENGAPPA, MUCKATIRA M	US

INT-CL (IPC): A61K 39/00; A61K 39/02
EUR-CL (EPC): C07K014/195

ABSTRACT:

A method is provided for the enhanced elaboration of leukotoxin from F. necrophorum, and subsequent production of an inactivated leukotoxoid ruminant animal vaccine against F. necrophorum infection and consequent liver abscesses and/or foot rot in such animals. The method involves forming a culture of F. necrophorum bacteria in growth media, allowing the bacteria to grow therein and to simultaneously elaborate leukotoxin in a supernate; the culturing is preferably carried out at a temperature of from about 35 DEG -41 DEG C., a pH of from about 6.5-8, and for a period of from about 4-9 hours. At the end oil of the culturing, bacterial growth and leukotoxin elaboration are terminated, preferably by separating the leukotoxin supernate, whereupon the vaccine is produced by inactivation of at least the supernate.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 3. Document ID: US 4152414 A

L20: Entry 3 of 9 File: EPAB May 1, 1979

PUB-NO: US004152414A
DOCUMENT-IDENTIFIER: US 4152414 A
TITLE: Combination vaccine for swine dysentery and method of use

PUBN-DATE: May 1, 1979

INVENTOR-INFORMATION:

NAME	COUNTRY
GLOCK, ROBERT D	US
GOODNOW, ROBERT A	US
HARRIS, DELBERT L	US
KINYON, JOANN M	US

INT-CL (IPC): A61K 39/02
EUR-CL (EPC): A61K039/02; A61K039/114

ABSTRACT:

A combination vaccine for increasing the resistance of swine to dysentery infection comprises killed cells of a virulent isolate of Treponema hyodysenteriae in combination with concentrated killed cells of Bacteroides vulgatus, or Fusobacterium necrophorum, or mixtures thereof. This combination vaccine can be adapted for either oral or parenteral administration. For parenteral administration preferably only Bacteroides vulgatus is used in combination with Treponema hyodysenteriae. The oral vaccine is enteric-coated.

☐ 4. Document ID: DE 20005024 U1

L20: Entry 4 of 9 File: DWPI Jun 15, 2000

DERWENT-ACC-NO: 2000-401484
DERWENT-WEEK: 200035

TITLE: New sheep foot rot vaccine comprises a mixture of inactivated anaerobic bacteria, including Dichelobacter nodosus and Pophyromonas levii

PRIORITY-DATA: 2000DE-2005024 (March 17, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
DE 20005024 U1	June 15, 2000		008	A61K039/39

INT-CL (IPC): A61K 39/39

ABSTRACTED-PUB-NO: DE 20005024U
BASIC-ABSTRACT:

NOVELTY - Sheep foot rot vaccine comprising inactivated anaerobic bacteria is new.

DETAILED DESCRIPTION - Sheep foot rot vaccine comprises:

(a) 10 - 95 parts by volume (pbv) of a mixture comprising 108 - 1010 cells/ml of inactivated anaerobic bacteria selected from (i) Dichelobacter nodosus, Pophyromonas levii, Prevotella loeschii, P. denticola, P. oralis, Bacteroides stercoris, B. ureolyticus, Fusobacterium nucleatum and F. necrophorum;

(b) up to 0.2 pbv of an inactivating agent;

(c) 5 - 90 pbv of an oil adjuvant or 5 - 10 pbv of a mineral adjuvant; and

(d) up to 0.45 pbv of a preservative.

USE - For preventing infectious foot rot in sheep.

ADVANTAGE - The vaccine is highly effective, protecting 75 - 95% of sheep in a flock against infection (no concrete data given).

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 5. Document ID: SU 1816348 A3

L20: Entry 5 of 9 File: DWPI Aug 20, 1995

DERWENT-ACC-NO: 1996-159082
DERWENT-WEEK: 199616

TITLE: Vaccine for prevention of necro-bacteriosis in cattle - contains endotoxin and exotoxin from Fusobacterium necrophorum

INVENTOR: KARAVAEV YU, D; MUSAEV, A R ; SEMENOVA, I N

PRIORITY-DATA: 1991SU-4928887 (March 29, 1991)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
SU 1816348 A3	August 20, 1995		004	A61K039/00

INT-CL (IPC): A61K 39/00

ABSTRACTED-PUB-NO: SU 1816348A
BASIC-ABSTRACT:

A vaccine for prevention of necrobacteriosis (NB) in cattle contains an endotoxin from Fusobacterium necrophorum (FN), formalin, an adjuvant, physiological soln. (PS) and also FN exotoxin, which is a protein of mol.wt. 18000-20000 Da, is decomposed by 0.5% trypsin soln. after 30-40 min. resulting in the death of rabbits on i.v. injection after 14-16 hrs., and haemolysis of sheep, horse, and rabbit erythrocytes. The FN endotoxin protein has mol.wt. 12000-15000 Da, is stable on heating to 70-75deg.C for 30 min. and on incubation with 0.5% trypsin when s.c. injected to rabbits (0.5 ml), it produces a dermatonecr otic reaction and lysis of sheep, horse, and rabbit erythrocytes. The adjuvant consists of a light mineral oil and anhydrous lanolin in ratio (83-85):(15-17). The proportions of the components are (per 1 of vaccine): 55.0-60.0 FN exotoxin protein (mol.wt. 18000-20000 Da, in PS in quantity 5.5-6.0 mg%), 25.0-30.0 FN endotoxin protein (mol.wt. 12000-15000 Da in PS in quantity 2.5-3.0 mg.%), 3.0-4.0 formalin, 415.0-425.0, light mineral oil, 75.0-85.0 anhydrous lanolin, and PS up to 1 l.

ADVANTAGE - The vaccine gives protracted and intensive immunity to cattle and sheep against NB.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Drawi Desc | Image

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☐ 6. Document ID: ES 2149821 T3, WO 9400556 A1, AU 9346451 A, ZA 9304591 A, EP 648260 A1, US 5455034 A, US 5492694 A, EP 648260 A4, AU 675234 B, EP 648260 B1, MX 191869 B, DE 69329494 E

L20: Entry 6 of 9 File: DWPI Nov 16, 2000

DERWENT-ACC-NO: 1994-026196
DERWENT-WEEK: 200064

TITLE: Increased prodn. of leukotoxin from fusobacterium necrophorum - used in vaccine to prevent infection in ruminants

INVENTOR: CHENGAPPA, M M; NAGARAJA, T G

PRIORITY-DATA: 1993US-0078066 (June 18, 1993), 1992US-0905041 (June 26, 1992), 1994US-0333767 (November 3, 1994)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
ES 2149821 T3	November 16, 2000		000	C12P021/00
WO 9400556 A1	January 6, 1994	E	046	C12N001/00
AU 9346451 A	January 24, 1994		000	C12N001/00
ZA 9304591 A	April 27, 1994		045	C12N000/00
EP 648260 A1	April 19, 1995	E	000	C12N001/00
US 5455034 A	October 3, 1995		014	A61K039/00
US 5492694 A	February 20, 1996		014	A61K039/00
EP 648260 A4	March 27, 1996		000	C12N001/00
AU 675234 B	January 30, 1997		000	C12N001/20
EP 648260 B1	September 27, 2000	E	000	C12P021/00
MX 191869 B	April 29, 1999		000	A61K039/000
DE 69329494 E	November 2, 2000		000	C12P021/00

INT-CL (IPC): A61K 37/02; A61K 39/00; A61K 39/000; A61K 39/002; A61K 39/02; A61K 39/114; C12N 0/00; C12N 1/00; C12N 1/12 ; C12N 1/20; C12P 21/00; C12P 21/04

ABSTRACTED-PUB-NO: EP 648260B
BASIC-ABSTRACT:

The elaboration of leukotoxin from Fusobacterium necrophorum is enhanced by: (a) culturing a biotype A F. necrophorum (b) causing elaboration of leukotoxin into the supernatant by culturing at 35-41 deg.C an pH 6.5-8 for 4-10 hrs., and (c) terminating growth and prodn. of leukotoxin while preserving most of the leukotoxin.

Also claimed is a vaccine produced by inactivating the leukotoxin supernatant produced above.

USE/ADVANTAGE - The vaccine can be used to vaccinate ruminant animals. This vaccine prevents leukotoxin prodn. or inhibits its activity, so preventing the establishment of F. necrophorum infection, e.g. liver abcesses in cattle and sheep, and foot rot in cattle.
ABSTRACTED-PUB-NO:

US 5455034A EQUIVALENT-ABSTRACTS:

The elaboration of leukotoxin from Fusobacterium necrophorum is enhanced by: (a) culturing a biotype A F. necrophorum (b) causing elaboration of leukotoxin into the supernatant by culturing at 35-41 deg.C an pH 6.5-8 for 4-10 hrs., and (c) terminating growth and prodn. of leukotoxin while preserving most of the leukotoxin.

Also claimed is a vaccine produced by inactivating the leukotoxin supernatant produced above.

USE/ADVANTAGE - The vaccine can be used to vaccinate ruminant animals. This vaccine prevents leukotoxin prodn. or inhibits its activity, so preventing the establishment of F. necrophorum infection, e.g. liver abcesses in cattle and sheep, and foot rot in cattle.

Vaccine is claimed, produced by (a) forming a culture of a biotype A strain of F. necrophorum bacteria in growth media, (b) growing the bacteria to enhance leukotoxin in the supernatant by culturing at 35-41 deg.C, pH 0.5-8 for 4-10 hours, and (c) forming the vaccine by inactivating at least the leukotoxin supernatant at the end of the culture.

USE/ADVANTAGE - As a vaccine against F. necrophorum infection in ruminants, and consequent liner abscesses and/or foot rot. Suitable ruminants include cattle and sheep. Elaboration of leukotoxin is enhanced.

US 5492694A

A method of enhancing the elaboration of leukotoxin from F. necrophorum, comprising the steps of: forming a culture of a biotype A strain of F. necrophorum bacteria in growth media; causing said bacteria to grow in said culture, and to elaborate leukotoxin in a supernate, including the steps of culturing at a temperature of from about 35-41 deg. C and a pH of from about 6.5-8 for a period of from about 4-10 hours; and terminating said bacterial growth and leukotoxin elaboration at the end of said period, while preserving a substantial proportion of the elaborated leukotoxin.

WO 9400556A

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 7. Document ID: US 6132709 A, EP 460480 A, AU 9178177 A, CA 2043932 A, NZ 238378 A, EP 460480 A3, AU 644440 B, EP 460480 B1, DE 69116606 E, ES 2084057 T3, IE 72181 B

L20: Entry 7 of 9 File: DWPI Oct 17, 2000

DERWENT-ACC-NO: 1991-362990
DERWENT-WEEK: 200054

TITLE: Treatment of foot rot and liver necrosis - in cattle and sheep, with a Fusobacterium necrophorum bacterin

INVENTOR: BERG, J N

PRIORITY-DATA: 1990US-0534894 (June 7, 1990), 1992US-0825465 (January 24, 1992)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 6132709 A	October 17, 2000		000	A01N063/00
EP 460480 A	December 11, 1991		000	
AU 9178177 A	December 12, 1991		000	
CA 2043932 A	December 8, 1991		000	
NZ 238378 A	December 23, 1992		000	A01N063/00
EP 460480 A3	April 1, 1992		000	
AU 644440 B	December 9, 1993		000	A61K039/114
EP 460480 B1	January 24, 1996	E	012	A61K035/74
DE 69116606 E	March 7, 1996		000	A61K035/74
ES 2084057 T3	May 1, 1996		000	A61K035/74
IE 72181 B	March 26, 1997		000	A61K035/74

INT-CL (IPC): A01N 63/00; A61K 35/74; A61K 39/11; A61K 39/114; C12P 1/04

ABSTRACTED-PUB-NO: EP 460480A
BASIC-ABSTRACT:

Treating cattle and sheep to prevent foot rot and/or liver necrosis comprises admin. of a fusobacterium necrophorum bacteria which is a beta-propiolactone inactivated F. necrophorum isolate to the animal being treated.

Also claimed are a process for the prodn. of the bacteria by isolating a virulent strain of F necrophorum and inactivating it with beta-propiolactone and a

beta-propiolactone inactivated F. necrophonum isolate (bacteria).

The bacteria specifically includes an adjuvant and has been treated to remove any residual beta-propiolactone present, pref. by hydrolysis. The adjuvant is then added.

USE/ADVANTAGE - The bacteria isolates are used to mfr. prods. for treating cattle and heep to prevent foot rot and/or liver necrosis. Better protection against Fusobacterium diseases is provided than with known treatments. Such diseases include liver abscess, calf diphtheria and interdigital dermatitis. Admin. of a vaccine contg. the bacteria is parenteral in a series of at least two injections, pref. by subcutaneous injection.
ABSTRACTED-PUB-NO:

EP 460480B EQUIVALENT-ABSTRACTS:

Use of fusobacterium necrophorum isolates for the manufacture of a bacterin for treating cattle and sheep to prevent foot rot.

US 6132709A

Treating cattle and sheep to prevent foot rot and/or liver necrosis comprises admin. of a fusobacterium necrophorum bacteria which is a beta-propiolactone inactivated F. necrophorum isolate to the animal being treated.

Also claimed are a process for the prodn. of the bacteria by isolating a virulent strain of F necrophorum and inactivating it with beta-propiolactone and a beta-propiolactone inactivated F. necrophonum isolate (bacteria).

The bacteria specifically includes an adjuvant and has been treated to remove any residual beta-propiolactone present, pref. by hydrolysis. The adjuvant is then added.

USE/ADVANTAGE - The bacteria isolates are used to mfr. prods. for treating cattle and heep to prevent foot rot and/or liver necrosis. Better protection against Fusobacterium diseases is provided than with known treatments. Such diseases include liver abscess, calf diphtheria and interdigital dermatitis. Admin. of a vaccine contg. the bacteria is parenteral in a series of at least two injections, pref. by subcutaneous injection.

☐ 8. Document ID: RO 101801 A

L20: Entry 8 of 9 File: DWPI Jul 30, 1991

DERWENT-ACC-NO: 1993-025083
DERWENT-WEEK: 199303

TITLE: Preparing vaccine for infectious foot rot of sheet - by culturing fusobacterium-necrophorum on protein-based substrate

INVENTOR: CHLURCLU, G C; CONSTANTLNESCU, M C ; POP, O T ; VERDES, N

PRIORITY-DATA: 1988RO-0135828 (November 11, 1988)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
RO 101801 A	July 30, 1991		008	A61K039/40

INT-CL (IPC): A61K 39/114; A61K 39/40

ABSTRACTED-PUB-NO: RO 101801A
BASIC-ABSTRACT:

Vaccine against infectious foot-dermatitis of cattle is prepd. as follows:

Fusobacterium necrophorum bacteria registered under No 32/1977 at the collection ICVB ''paskur'' /ASI are cultured on a substrate contg. (all in %-s): 12 enzyme hydrolysed edoeine, 10 yeast extract, 30 meat extract, 1 peptone, 0.5 cysteine-hydro-chloride, 0.2 glycerol; at pH 7.4 and 37 deg. C, for 6 hrs. in a kinetic, or for 48-72 hrs. in a static system. Completed culture is 20 x by tangential ultra filtration or 40 x by centrifuging. The antigenic mass is disintegrated for 10 mins. in a static system or in a flow-through disintegrator of 300 W power at a rate of 3 l-s/hr, for 3 hrs. Deactivation by 0.5% formaldehyde follows. The prod. is adsorbed by 20% aluminium-hydroxid e and a final concn. of 8-10 mg/l protein is reached by adding culturing liq. produce

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 9. Document ID: US 4152414 A

L20: Entry 9 of 9 File: DWPI May 1, 1979

DERWENT-ACC-NO: 1979-38889B
DERWENT-WEEK: 197920

TITLE: Vaccine for increasing resistance to swine dysentery infection - comprising killed cells of treponema hyodysenteriae and Bacteroides vulgatus or Fusobacterium necrophorum

INVENTOR: GLOCK, R D; GOODNOW, R A ; HARRIS, D L

PRIORITY-DATA: 1978US-0935000 (August 18, 1978), 1979US-0016623 (March 1, 1979)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 4152414 A	May 1, 1979		000	

ABSTRACTED-PUB-NO: US 4152414A
BASIC-ABSTRACT:

Oral prepn. for increasing the resistance of swine to dysentery infection comprises enteric-coated pellets contg. concd. Kiled cells of a virulent isolates of Treponema hyodysenteriae with 0.25-2 pts. of concd. killed cells of BActeroides vulgatus or Furobacterium necrophorium or their mixts.

The vaccine has an enhanced action in increasing the resistance of swine to dysentery infection, and can be used either as an oral or parenteral vaccine or may be further adapted for intramuscular injection. The oral vaccine can be mixed with feed.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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Term	Documents
(19 NOT (14 OR 10 OR 7)).JPAB,EPAB,DWPI.	9

Display 25 Documents, starting with Document: 9

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☐ 1. Document ID: JP 2000072667 A

L25: Entry 1 of 8 File: JPAB Mar 7, 2000

PUB-NO: JP02000072667A
DOCUMENT-IDENTIFIER: JP 2000072667 A
TITLE: ORALLY ADMINISTRATIVE AGENT FOR TREATING COLITIS

PUBN-DATE: March 7, 2000

INVENTOR-INFORMATION:
NAME COUNTRY
MIYASAKA, TOSHIO

INT-CL (IPC): A61K 31/19; A61K 9/28

ABSTRACT:

PROBLEM TO BE SOLVED: To obtain the subject orally administrative agent containing a short- chain fatty acid (derivative) as active ingredient and effective for treating colitis caused by antibiotic administration, and also so designed as to be prevented from emitting offensive odor (oxidative rancidity) unique to dose of the short- chain fatty acid and from being absorbed through the stomach and small intestine when orally administered.

SOLUTION: This orally administrative agent for treating colitis is obtained by enteric coating of a medicinal mixture containing, as active ingredient, a short-chain fatty acid or derivative thereof such as butyric acid, sodium butyrate or butyrate ester. When needed, prior to the enteric coating, the medicinal mixture is subjected to film-undercoating or, after the enteric coating, the mixture is made into a sugarcoated tablet form or encapsulated.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw. Desc | Image

☐ 2. Document ID: US 5840860 A

L25: Entry 2 of 8 File: EPAB Nov 24, 1998

PUB-NO: US005840860A
DOCUMENT-IDENTIFIER: US 5840860 A
TITLE: Fatty acid delivery system comprising a hydrolyzable bond

PUBN-DATE: November 24, 1998

INVENTOR-INFORMATION:

NAME	COUNTRY
ANNISON, GEOFFREY	SG
TOPPING, DAVID L	AU
ILLMAN, RICHARD J	AU

INT-CL (IPC): C07H 1/00
EUR-CL (EPC): A61K031/19; A61K031/20, A61K047/48 , A21D002/16 , A23L001/30

ABSTRACT:

PCT No. PCT/AU94/00713 Sec. 371 Date Sep. 5, 1996 Sec. 102(e) Date Sep. 5, 1996 PCT Filed Nov. 17, 1994 PCT Pub. No. WO95/13801 PCT Pub. Date May 26, 1995Delivery to the colon of fatty acids especially Short Chain Fatty Acids (SCFA) can be effected by covalently linking SCFA to a carrier that is preferably a form of carbohydrate, by an ester link. The SCFA is protected by its link with the carbohydrate through the small intestine, and where the carbohydrate is digestible in the small intestine such as a digestible starch, the starch can also be protected from digestion in the small intestine by the substitution. Levels of SCFA such as acetate, propionate and butyrate may be elevated to have beneficial effects in the prevention of colonic disorders such as rectal cancer, diverticulities, colitis, diarrhea and constipation.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

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☐ 3. Document ID: WO 9513801 A1

L25: Entry 3 of 8 File: EPAB May 26, 1995

PUB-NO: WO009513801A1
DOCUMENT-IDENTIFIER: WO 9513801 A1
TITLE: FATTY ACID DELIVERY SYSTEM

PUBN-DATE: May 26, 1995

INVENTOR-INFORMATION:

NAME	COUNTRY
ANISSON, GEOFFREY	SG
TOPPING, DAVID	AU
ILLMAN, RICHARD	AU

INT-CL (IPC): A61K 31/19; A61K 47/36; A61K 47/38
EUR-CL (EPC): A61K031/19; A61K031/20, A21D002/16 , A23L001/30 , A61K047/48

ABSTRACT:

Delivery to the colon of fatty acids especially Short Chain Fatty Acids (SCFA) can be effected by covalently linking SCFA to a carrier that is preferably a form of carbohydrate, by an ester link. The SCFA is protected by its link with the carbohydrate through the small intestine, and where the carbohydrate is digestible in the small intestine such as a digestible starch, the starch can also be protected from digestion in the small intestine by the substitution. Levels of SCFA such as acetate, propionate and butyrate may be elevated to have beneficial effects in the prevention of colonic disorders such as rectal cancer, diverticulitis, colitis, diarrhea and constipation.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 4. Document ID: US 5266315 A

L25: Entry 4 of 8 File: EPAB Nov 30, 1993

PUB-NO: US005266315A
DOCUMENT-IDENTIFIER: US 5266315 A
TITLE: Composite for Clostridium difficile diarrhea and pseudomembranous colitis

PUBN-DATE: November 30, 1993

INVENTOR-INFORMATION:

NAME	COUNTRY
TAGUCHI, NOBUHIRO	JP
FUJITA, ITSUKI	JP

INT-CL (IPC): C12N 1/20
EUR-CL (EPC): A61K035/74; A61K035/74, A61K038/14

ABSTRACT:

A preventive and curative medical composition for Clostridium difficile diarrhea and psedomembranous colitis, which composition comprises cells or spores of a butyric acid bacterium and Vancomycin.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 5. Document ID: EP 456418 A2

L25: Entry 5 of 8 File: EPAB Nov 13, 1991

PUB-NO: EP000456418A2
DOCUMENT-IDENTIFIER: EP 456418 A2
TITLE: Treatment of clostridium difficile diarrhoea and pseudomembranous colitis.

PUBN-DATE: November 13, 1991

INVENTOR-INFORMATION:

NAME	COUNTRY
FUJITA, ITSUKI	JP
TAGUCHI, NOBUHIRO	JP

INT-CL (IPC): A61K 35/74; A61K 37/02
EUR-CL (EPC): A61K035/74; A61K035/74, A61K038/14

ABSTRACT:

A preventive and/or curative pharmaceutical composition for Clostridium difficile diarrhoea and pseudomembranous colitis comprising cells or spores of a butyric acid bacterium and an anti bacterial agent, for example vancomycin. The butyric acid bacterium may be Clostridium butyricum.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

☐ 6. Document ID: EP 446069 A1

L25: Entry 6 of 8 File: EPAB Sep 11, 1991

PUB-NO: EP000446069A1
DOCUMENT-IDENTIFIER: EP 446069 A1
TITLE: Treatment of Clostridium difficile diarrhoea and pseudomembranous colitis.

PUBN-DATE: September 11, 1991

INVENTOR-INFORMATION:

NAME	COUNTRY
KUROIWA, TOYOAKI	JP
TAGUCHI, NOBUHIRO	JP

INT-CL (IPC): A61K 35/74
EUR-CL (EPC): A61K035/74; A61K035/74

ABSTRACT:

CHG DATE=19990617 STATUS=O> A preventative and/or curative pharmaceutical composition for Clostridium difficile diarrhoea and pseudomembranous colitis comprises cells or spores of a butyric acid bacterium and a saccharide. The butyric acid bacterium may be Clostridium butyricum and the saccharide may be a polysaccharide such as raffinose and/or starch.

☐ 7. Document ID: KR 149672 B1, EP 456418 A, JP 04013628 A, HU 61464 T, EP 456418 A3, US 5266315 A, EP 456418 B1, DE 69122298 E, ES 2091290 T3, JP 2961184 B2

L25: Entry 7 of 8 File: DWPI Oct 15, 1998

DERWENT-ACC-NO: 1991-334365
DERWENT-WEEK: 200026

TITLE: Treating Clostridium difficile diarrhoea and pseudo:membranous colitis - with compsn. comprising cells or spores of butyric acid bacterium and antibiotic

INVENTOR: FUJITA, I; TAGUCHI, N

PRIORITY-DATA: 1990JP-0117267 (May 7, 1990)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
KR 149672 B1	October 15, 1998		000	A61K039/08
EP 456418 A	November 13, 1991		000	
JP 04013628 A	January 17, 1992		000	
HU 61464 T	January 28, 1993		000	A61K031/44
EP 456418 A3	January 20, 1993		000	
US 5266315 A	November 30, 1993		005	C12N001/20
EP 456418 B1	September 25, 1996	E	005	A61K038/14
DE 69122298 E	October 31, 1996		000	A61K038/14
ES 2091290 T3	November 1, 1996		000	A61K038/14
JP 2961184 B2	October 12, 1999		008	A61K035/74

INT-CL (IPC): A61K 31/00; A61K 31/44; A61K 31/70; A61K 35/66; A61K 35/74; A61K 38/00; A61K 38/14; A61K 39/08; C12N 1/20 ; A61K 35/74; A61K 38/14; A61K 35/74; A61K 38/14

ABSTRACTED-PUB-NO: EP 456418A
BASIC-ABSTRACT:

The amt. of antibacterial agent is 1-10000 mg per 10 power 8 CFU of the butyric acid bacterium cells or spores. The antibacterial agent and bacterial cells/spores may be administered sequentially or as a combined prepn.

USE/ADVANTAGE - Low toxicity and minimal impairment of intestinal microflora. The Clostridium butyricum cells/spores prevent recurrence of the disease, which often takes place when vancomycin alone is administered. Admin. is oral. (Previously notified in Week 9146)
ABSTRACTED-PUB-NO:

EP 456418B EQUIVALENT-ABSTRACTS:

A pharmaceutical and/or veterinary composition comprising cells or spores of a butyric acid bacterium and vancomycin.

US 5266315A

Medical compsn. for the treatment of clostridium difficile diarrhoea and

pseudomembranous colitis, comprises a combination of cells or spores of Clostridium butyricum and vancomycin.

Amt. of vancomycin used is 1-10,000 mg per 100,000,000 CFU of the cells/spores. Cells/spores are used in amt. 1,000,000-1,000,000,000 CFU per kg. human body wt.

ADVANTAGE - Is preventive and curative, and has very low toxicity.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

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☐ 8. Document ID: KR 145553 B1, EP 446069 A, HU 61478 T, EP 446069 B1, DE 69100314 E, JP 06056679 A, RO 106195 B1, JP 2961182 B2

L25: Entry 8 of 8 File: DWPI Aug 17, 1998

DERWENT-ACC-NO: 1991-268907
DERWENT-WEEK: 200021

TITLE: Compsn. of cells or spores of butyric acid bacterium and saccharide - for treatment of clostridium difficle diarrhoea and pseudo-membranous colitis

INVENTOR: KUROIWA, T; TAGUCHI, N

PRIORITY-DATA: 1990JP-0056596 (March 9, 1990)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
KR 145553 B1	August 17, 1998		000	A61K035/74
EP 446069 A	September 11, 1991		012	
HU 61478 T	January 28, 1993		000	A61K035/66
EP 446069 B1	September 1, 1993	E	011	A61K035/74
DE 69100314 E	October 7, 1993		000	A61K035/74
JP 06056679 A	March 1, 1994		008	A61K035/74
RO 106195 B1	March 31, 1993		000	A61K039/08
JP 2961182 B2	October 12, 1999		008	A61K035/74

INT-CL (IPC): A61K 31/00; A61K 31/70; A61K 35/66; A61K 35/74; A61K 39/08; A61K 47/26; A61K 47/36; A61K 31/70; A61K 35/74

ABSTRACTED-PUB-NO: EP 446069A
BASIC-ABSTRACT:

The butyric acid bacterium is pref. Clostridium butyriium. The compsn. contains 1-10,000 mg of saccharide based on 10 power -8 CFU of the cells or spores of the butyric acid bacterium. The saccharide is a polysaccharide power esp. raffinose and/or starch.

USE/ADVANTAGE - For treating Clostridium difficile diarrhoea and pseudomembranous colitis (claimed).

In an example, after the start of antibacterial therapy using penicillins and cephalosporins in patients who had no digestive tract diseases, the patients were divided into a first gp. subjected to admin. of 1-3 doses each of 1g of ''Miya BM'' (a blend of 40mg of ground Clostridium butyricium MIYA/R1 588 (CbM) cells with 960 mg of corn starch and contg. 10 power -8 CFU of CbM spores per g) as a powdered CbM medicine daily, and a second gp. who were not subjected to admin. Faeces were examined for traces of Clostridium difficile and its toxin. The results show that Clostridium difficile or its toxin was detected in 5% of the

results show that Clostridium difficile or its toxin was detected in 5% of the patients in the CbM admin. gp. compared with 30% of the patients in the gp. not administered wth CbM.
ABSTRACTED-PUB-NO:

EP 446069B EQUIVALENT-ABSTRACTS:

A pharmaceutical composition comprising cells or spores of a butyric acid bacterium and a polysaccharide.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

Generate Collection

Term	Documents
(3 WITH 21).JPAB,EPAB,DWPI.	8

Display 25 Documents, starting with Document: 8

Display Format: REV Change Format

WEST

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Search Results - Record(s) 1 through 4 of 4 returned.

☐ 1. Document ID: US 5214066 A

L30: Entry 1 of 4 File: EPAB May 25, 1993

PUB-NO: US005214066A
DOCUMENT-IDENTIFIER: US 5214066 A
TITLE: Method for producing an animal model for inflammatory bowel disease including ulcerative colitis

PUBN-DATE: May 25, 1993

INVENTOR-INFORMATION:

NAME	COUNTRY
SZABO, SANDOR	US

INT-CL (IPC): A01N 43/36; A61K 31/40
EUR-CL (EPC): A61K031/16; A61K031/19, A61K031/40

ABSTRACT:

A new animal model for Inflammatory Bowel Disease, including idiopathic ulcerative colitis and Chron's disease, as well as methods for problems such as an animal, is provided. Chronic ulcerative condition is induced by topical administration of a sulfhydryl blocker, such as N-ethylmaleimide or iodoacetamide, to the colon. The new animal model is useful for studying the pathogenesis of chronic ulcerative disease, and prevention and treatment thereof, and for evaluating drugs suspected of being useful in the treatment of same.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

☐ 2. Document ID: AU 200123376 A, WO 200147533 A2

L30: Entry 2 of 4 File: DWPI Jul 9, 2001

DERWENT-ACC-NO: 2001-418157
DERWENT-WEEK: 200164

TITLE: Inhibiting nuclear factor-kappa-B activity in a cell for treating asthma, lupus, scleroderma, cancer, psoriasis, inflammation, comprises contacting the cell with an inhibitor of glycogen synthase kinase-3

INVENTOR: HOEFLICH, K; LUO, J ; WOODGETT, J

PRIORITY-DATA: 1999US-0172064 (December 23, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 200123376 A	July 9, 2001		000	A61K033/14
WO 200147533 A2	July 5, 2001	E	032	A61K033/14

INT-CL (IPC): A01K 67/027; A61K 33/14; A61K 45/00; A61P 29/00; A61P 35/00

ABSTRACTED-PUB-NO: WO 200147533A
BASIC-ABSTRACT:

NOVELTY - Inhibiting (M1) activity of nuclear factor-kappa-B (NF-kB) in a cell, involves contacting the cell with an inhibitor of glycogen synthase kinase-3 (GSK-3).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a non-human transgenic animal model (I) for GSK-3 gene function which has a defect in GSK-3 function;

(2) screening (M2) for biologically active agents that modulate GSK-3b function, involving combining a candidate agent with a non-human transgenic animal comprising a knockout of an GSK-3b gene or an exogenous and stably transmitted mammalian GSK-3b gene sequence, and determining the effect of the agent on GSK-3b function; and

(3) screening (M3) biologically active agents for the specificity of action on GSK-3b function, involving combining a candidate agent with a non-human transgenic animal comprising a knockout of an GSK-3b gene or an exogenous and stably transmitted mammalian GSK-3b gene sequence, and determining the effect of the agent on GSK-3b function as compared to an animal comprising normal GSK-3b function.

ACTIVITY - Antiasthmatic; neuroprotective; antipsoriatic; antiarthritic; antiinflammatory; cytostatic; dermatological; immunosuppressive; antithyroid; antiulcer; vasotropic.

MECHANISM OF ACTION - Inhibitor of NF-kB activity (claimed). No supporting data is given.

USE - M1 is useful for modulating the activity of NF-kB activity (claimed). M1 is useful for treating asthma, systemic lupus erythematosus (SLE), scleroderma, various forms of vasculitis, inflammatory autoimmune myositis, autoimmune thyroiditis, multiple sclerosis, inflammatory arthritis, inflammatory bowel diseases including Crohn's disease and ulcerative colitis, psoriasis, systemic shock and hyperproliferative disorders such as arthritis, inflammation, cancer etc. A non-human transgenic animal model (I) which has a defect in GSK-3 function is useful for assessing the effect of a compound on GSK-3 inhibitor cells, and for the determination of pathways relating to GSK-3.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 3. Document ID: JP 08188600 A

L30: Entry 3 of 4 File: DWPI Jul 23, 1996

DERWENT-ACC-NO: 1996-388604
DERWENT-WEEK: 199639

TITLE: New antibodies inhibiting rat's II-type phospholipase A2 and its fragments - isolating antibody from rat's platelet and releasing phospholipase A2 bound to cells, used in treating e.g. asthma, psoriasis, etc..

PRIORITY-DATA: 1994JP-0340005 (December 29, 1994)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 08188600 A	July 23, 1996		015	C07K016/40

INT-CL (IPC): C07K 16/40; C12N 5/10; C12N 15/02; C12P 21/08; C12P 21/08; C12R 1/91

ABSTRACTED-PUB-NO: JP08188600A
BASIC-ABSTRACT:

An antibody having the following properties and its fragment are new: (1) inhibiting II-type phospholipase A2 (PLA2) isolated from rat's platelet, and (2) releasing the PLA2 bound to cells. Also claimed is a murine monoclonal antibody (MAb) 3A1 or 2R7 produced by a hybridoma 3A1 (FERM P-14654) or 2F7 (FERM P-14653), respectively, or its reduced alkylated deriv. or its fragment. Also claimed is a hybridoma 3A1 or 2F7.

MAb which does not inhibit PLA2 of human, rhesus monkey, dog, rabbit, mouse and cat. Reduced alkylated antibody or its fragment which is prepd. by reducing he S-S linkage between H- and H-chains and/or H- and L-chain and then alkylating with iodoacetonitrile.

USE/ADVANTAGE - The antibody or its fragment (F(ab')2, Fab, or Fab' derived from whole molecule by digestion with trypsin, papain or pepsin) may be used in development of drugs for diseases (e.g. myocardial infarction, cerebral infarction, acute nephropathy, asthma, chronic rheumarthritis, osteoarthritis, septic shock, pancreatitis, psoriasis, multiple organ failure (MOF), acute respiratory distress syndrome (ARDS), Crohn disease and chronic ulcerative colitis, uveitis, respiratory disturbance syndrome (RDS) in new born, bronchopulmonary dysplasia (BFD)) partially or totally mediated by PLA2, using pathogenic rat models. The antibody strongly reacts with rat's PLA2 to completely inhibit its activity at a concn. of 3 micro g/ml (polyclonal). IC50 = 0.1 micro g/ml.

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 4. Document ID: US 5214066 A

L30: Entry 4 of 4 File: DWPI May 25, 1993

DERWENT-ACC-NO: 1993-181825
DERWENT-WEEK: 199322

TITLE: Producing animal model for inflammatory bowel disease - by topical admin. to colonic mucosa of a sulphydryl blocker

INVENTOR: SZABO, S

PRIORITY-DATA: 1990US-0510229 (April 18, 1990)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 5214066 A	May 25, 1993		007	A01N043/36

INT-CL (IPC): A01N 43/36; A61K 31/40

ABSTRACTED-PUB-NO: US 5214066A
BASIC-ABSTRACT:

Prodn. of mammalian idiopathic inflammatory bowel disease in a non-primate laboratory animal comprises topically administering to the colonic mucosa of the animal a compsn. comprising a sulphydryl blocker in an amount effective to produce colonic lesions.

USE - There is provided an animal model for inflammatory bowel disease, including idiopathic ulcerative colitis and Crohn's disease. The model is useful for studying the pathogenesis of chronic ulcerative disease and evaluating protective drugs.

WEST

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Search Results - Record(s) 1 through 1 of 1 returned.

☐ 1. Document ID: US 6291656 B1, WO 9921865 A1, AU 9892773 A

L38: Entry 1 of 1 File: DWPI Sep 18, 2001

DERWENT-ACC-NO: 1999-312936
DERWENT-WEEK: 200157

TITLE: New tricyclic erythromycin derivatives useful as broad spectrum antibiotics for treating bacterial and protozoal infections in mammals, fish or birds

INVENTOR: WU, Y

PRIORITY-DATA: 1997US-0063161 (October 29, 1997), 1999US-0341888 (July 15, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 6291656 B1	September 18, 2001		000	C07H017/08
WO 9921865 A1	May 6, 1999	E	029	C07H017/08
AU 9892773 A	May 17, 1999		000	C07H017/08

INT-CL (IPC): A61K 31/70; C07H 17/08

ABSTRACTED-PUB-NO: US 6291656B
BASIC-ABSTRACT:

NOVELTY - Tricyclic erythromycin derivatives (I) and their salts are new.

DETAILED DESCRIPTION - Tricyclic erythromycin derivatives of formula (I) and their salts are new.

R1, R2, R4 = H or 1-12C alkyl, where one or two carbons of the alkyl are optionally replaced by O, S or N, and are optionally substituted by 1-3 of C(O)OR6, OR6, 1-10C alkanoyl, halo, nitro, cyano, R6, R8, NR6R7, SR6, SOR6, SO2R6 or SO2NR6R7;

R3 = H, R7, C(O)R7, C(O)R8, C(O)OR7, C(O)OR8 or (CR6R7)mR8;

m = 0-6;

R5 = H, C(O)R8 or 1-18C alkanoyl, where in the alkyl portion of the alkanoyl, one or two carbons may optionally be replaced by O, S or N;

R6, R7 = H or 1-12C alkyl; and

R8 = 4-10 membered heterocyclyl or 6-10C aryl, where the heterocyclyl and aryl groups are optionally substituted by 1-3 of 4-10 membered heterocyclyl, 6-10C aryl, NHC(O)R6, NHC(O)NR6R7, C(O)OR6, OR6, C(O)R6, halo, nitro, cyano, R6, NR6R7, SR6, S(O)R6, SO2R6 or SO2NR6R7.

An INDEPENDENT CLAIM is also included for the preparation of (I).

ACTIVITY - Antibacterial; antiprotozoal. Assays of the activity of compounds (I) against bacterial and protozoal pathogens in humans and animals are described but no results are given.

MECHANISM OF ACTION - None given.

USE - Broad spectrum antibiotics for treating gram positive and gram negative bacterial and protozoal infections in mammals, fish or birds. These include pneumonia, otitis media, sinusitus, bronchitis, tonsillitis, and mastoiditis caused by Streptococcus pneumoniae, Haemophilus influenzae, Moraxelia catarrhalis, Staphylococcus aureus, or Peptostreptococcus spp.; pharynigitis, rheumatic fever, and glomerulonephritis caused by Streptococcus pyogenes, Groups C and G streptococci, Clostridium diptheriae, or Actinobacillus haemolyticum, respiratory tract infections caused by Mycoplasma pneumoniae, Legionelia pneumophila, Streptococcus pneumoniae, Haemophilus influenzae, or Chlamydia pneumoniae; uncomplicated skin and soft tissue infections, abscesses and osteomyelitis, and puerperal fever caused by Staphylococcus aureus, coagulase-positive staphylococci (e.g. S. epidermidis and S. hemolyticus), Streptococcus pyogenes , Streptococcus agalactiae, Streptococcal groups C-F (minute-colony streptococci), viridans streptococci, Corynebacterium minutissimum, Clostridium spp., or Bartonella henselae; uncomplicated acute urinary tract infections caused by Staphylococcus saprophyticus or Enterococcus spp.; urethritis and cervicitis; and sexually transmitted diseases caused by Chlamydia trachomatis, Haemophilus ducreyi, Treponema pallidum, Ureaplasma urealyticum, or Neiserria gonorrheae; toxin diseases caused by S. aureus (food poisoning and Toxic shock syndrome), or Groups A, B, and C streptococci; ulcers caused by Helicobacter pylori, systemic febrile syndromes related to infection by Borrelia recurrentis; lyme disease caused by Borrelia burgdorferi; conjunctivitis, keratitis, and dacrocystitis caused by C. trachomatis, N. gonorrhoeae, S. aureus, S. pneumoniae, S. pyogenes, H. influenzae, or Listeria spp.; disseminated Mycobacterium avium complex (MAC) disease caused by Mycobacteriurn avium, or Mycobacterium intracellulare; gastroenteritis caused by Campylobacter jejuni; intestinal protozoa caused by Cryptosporidium spp.; odontogenic infection caused by viridans streptococci; persistent cough caused by Bordetelia pertussis; gas gangrene caused by Clostridium perfringens or Bacteroides spp.; and atherosclerosis caused by Helicobacter pylori or Chlamydia pneumoniae. Bacterial infections and protozoa infections and disorders related to such infections that may be treated or prevented in animals include the following: bovine respiratory disease caused by P. haem, P. multocida, Mycoplasma bovis, or Bordetella spp.; cow enteric disease caused by E. coli or protozoa (i.e., coccidia, cryptosporidia, etc.); dairy cow mastitis caused by Staph. aureus, Strep. uberis, Strep. agalactiae, Strep. dysgalactiae, Klebsiella spp., Corynebacterium, or Enterococcus spp.; swine respiratory disease caused by A. pleuro, P. multocida, or Mycoplasma spp.; swine enteric disease related to infection by E. coli, Lawsonia intracellularis, Salmonella, or Serpulina hyodyisinteriae; cow footrot caused by Fusobacterium spp.; cow metritis caused by E. coli; cow hairy warts caused by Fusobacterium necrophorum or Bactercides nodosus; cow pink-eye caused by Moraxelia bows; cow premature abortion caused by protozoa (i.e. neosporium); urinary tract infection in dogs and cats caused by E. coli; skin and soft tissue infections in dogs and cats caused by Staph. epidermidis, Staph. intermedius, coagulase neg. Staph. or P. multocida; and dental or mouth infections in dogs and cats caused by Alcaligenes spp., Bacteroides spp., Clostridium spp., Enterobacter spp., Eubacterium, Peptostreptococcus, Porphyromonas, or Prevotelia. Other infections which may be treated or prevented using (I) are found in J.P. Sanford et al., The Sanford Guide to Antimicrobial Therapy, 26th Edition, (Antimicrobial Therapy, Inc. 1996).

Radiolabelled compounds (I) may be used as research or diagnostic tools.
ABSTRACTED-PUB-NO:

WO 9921865A EQUIVALENT-ABSTRACTS:

NOVELTY - Tricyclic erythromycin derivatives (I) and their salts are new.

DETAILED DESCRIPTION - Tricyclic erythromycin derivatives of formula (I) and their salts are new.

R1, R2, R4 = H or 1-12C alkyl, where one or two carbons of the alkyl are optionally replaced by O, S or N, and are optionally substituted by 1-3 of C(O)OR6, OR6, 1-10C alkanoyl, halo, nitro, cyano, R6, R8, NR6R7, SR6, SOR6, SO2R6 or SO2NR6R7;

R3 = H, R7, C(O)R7, C(O)R8, C(O)OR7, C(O)OR8 or (CR6R7)mR8;

m = 0-6;

R5 = H, C(O)R8 or 1-18C alkanoyl, where in the alkyl portion of the alkanoyl, one or two carbons may optionally be replaced by O, S or N;

R6, R7 = H or 1-12C alkyl; and

R8 = 4-10 membered heterocyclyl or 6-10C aryl, where the heterocyclyl and aryl groups are optionally substituted by 1-3 of 4-10 membered heterocyclyl, 6-10C aryl, NHC(O)R6, NHC(O)NR6R7, C(O)OR6, OR6, C(O)R6, halo, nitro, cyano, R6, NR6R7, SR6, S(O)R6, SO2R6 or SO2NR6R7.

An INDEPENDENT CLAIM is also included for the preparation of (I).

ACTIVITY - Antibacterial; antiprotozoal. Assays of the activity of compounds (I) against bacterial and protozoal pathogens in humans and animals are described but no results are given.

MECHANISM OF ACTION - None given.

USE - Broad spectrum antibiotics for treating gram positive and gram negative bacterial and protozoal infections in mammals, fish or birds. These include pneumonia, otitis media, sinusitus, bronchitis, tonsillitis, and mastoiditis caused by Streptococcus pneumoniae, Haemophilus influenzae, Moraxelia catarrhalis, Staphylococcus aureus, or Peptostreptococcus spp.; pharynigitis, rheumatic fever, and glomerulonephritis caused by Streptococcus pyogenes, Groups C and G streptococci, Clostridium diptheriae, or Actinobacillus haemolyticum, respiratory tract infections caused by Mycoplasma pneumoniae, Legionelia pneumophila, Streptococcus pneumoniae, Haemophilus influenzae, or Chlamydia pneumoniae; uncomplicated skin and soft tissue infections, abscesses and osteomyelitis, and puerperal fever caused by Staphylococcus aureus, coagulase-positive staphylococci (e.g. S. epidermidis and S. hemolyticus), Streptococcus pyogenes , Streptococcus agalactiae, Streptococcal groups C-F (minute-colony streptococci), viridans streptococci, Corynebacterium minutissimum, Clostridium spp., or Bartonella henselae; uncomplicated acute urinary tract infections caused by Staphylococcus saprophyticus or Enterococcus spp.; urethritis and cervicitis; and sexually transmitted diseases caused by Chlamydia trachomatis, Haemophilus ducreyi, Treponema pallidum, Ureaplasma urealyticum, or Neiserria gonorrheae; toxin diseases caused by S. aureus (food poisoning and Toxic shock syndrome), or Groups A, B, and C streptococci; ulcers caused by Helicobacter pylori, systemic febrile syndromes related to infection by Borrelia recurrentis; lyme disease caused by Borrelia burgdorferi; conjunctivitis, keratitis, and dacrocystitis caused by C. trachomatis, N. gonorrhoeae, S. aureus, S. pneumoniae, S. pyogenes, H. influenzae, or Listeria spp.; disseminated Mycobacterium avium complex (MAC) disease caused by Mycobacteriurn avium, or Mycobacterium intracellulare; gastroenteritis caused by Campylobacter jejuni; intestinal protozoa caused by Cryptosporidium spp.; odontogenic infection caused by viridans streptococci; persistent cough caused by Bordetelia pertussis; gas gangrene caused by Clostridium perfringens or Bacteroides spp.; and atherosclerosis caused by Helicobacter pylori or Chlamydia pneumoniae. Bacterial infections and protozoa infections and disorders related to such infections that may be treated or prevented in animals include the following: bovine respiratory disease caused by P. haem, P. multocida, Mycoplasma bovis, or Bordetella spp.; cow enteric disease caused by E. coli or protozoa (i.e., coccidia, cryptosporidia, etc.); dairy cow mastitis caused by Staph.

aureus, Strep. uberis, Strep. agalactiae, Strep. dysgalactiae, Klebsiella spp., Corynebacterium, or Enterococcus spp.; swine respiratory disease caused by A. pleuro, P. multocida, or Mycoplasma spp.; swine enteric disease related to infection by E. coli, Lawsonia intracellularis, Salmonella, or Serpulina hyodyisinteriae; cow footrot caused by Fusobacterium spp.; cow metritis caused by E. coli; cow hairy warts caused by Fusobacterium necrophorum or Bactercides nodosus; cow pink-eye caused by Moraxelia bows; cow premature abortion caused by protozoa (i.e. neosporium); urinary tract infection in dogs and cats caused by E. coli; skin and soft tissue infections in dogs and cats caused by Staph. epidermidis, Staph. intermedius, coagulase neg. Staph. or P. multocida; and dental or mouth infections in dogs and cats caused by Alcaligenes spp., Bacteroides spp., Clostridium spp., Enterobacter spp., Eubacterium, Peptostreptococcus, Porphyromonas, or Prevotelia. Other infections which may be treated or prevented using (I) are found in J.P. Sanford et al., The Sanford Guide to Antimicrobial Therapy, 26th Edition, (Antimicrobial Therapy, Inc. 1996).

Radiolabelled compounds (I) may be used as research or diagnostic tools.

Term	Documents
(37 NOT (14 OR 25 OR 10 OR 7 OR 19)).JPAB,EPAB,DWPI.	1

WEST

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Search Results - Record(s) 1 through 4 of 4 returned.

☐ 1. Document ID: US 5214066 A

L43: Entry 1 of 4 File: EPAB May 25, 1993

PUB-NO: US005214066A
DOCUMENT-IDENTIFIER: US 5214066 A
TITLE: Method for producing an animal model for inflammatory bowel disease including ulcerative colitis

PUBN-DATE: May 25, 1993

INVENTOR-INFORMATION:

NAME	COUNTRY
SZABO, SANDOR	US

INT-CL (IPC): A01N 43/36; A61K 31/40
EUR-CL (EPC): A61K031/16; A61K031/19, A61K031/40

ABSTRACT:

A new animal model for Inflammatory Bowel Disease, including idiopathic ulcerative colitis and Chron's disease, as well as methods for problems such as an animal, is provided. Chronic ulcerative condition is induced by topical administration of a sulfhydryl blocker, such as N-ethylmaleimide or iodoacetamide, to the colon. The new animal model is useful for studying the pathogenesis of chronic ulcerative disease, and prevention and treatment thereof, and for evaluating drugs suspected of being useful in the treatment of same.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

☐ 2. Document ID: AU 200123376 A, WO 200147533 A2

L43: Entry 2 of 4 File: DWPI Jul 9, 2001

DERWENT-ACC-NO: 2001-418157
DERWENT-WEEK: 200164

TITLE: Inhibiting nuclear factor-kappa-B activity in a cell for treating asthma, lupus, scleroderma, cancer, psoriasis, inflammation, comprises contacting the cell with an inhibitor of glycogen synthase kinase-3

INVENTOR: HOEFLICH, K; LUO, J ; WOODGETT, J

PRIORITY-DATA: 1999US-0172064 (December 23, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 200123376 A	July 9, 2001		000	A61K033/14
WO 200147533 A2	July 5, 2001	E	032	A61K033/14

INT-CL (IPC): A01K 67/027; A61K 33/14; A61K 45/00; A61P 29/00; A61P 35/00

ABSTRACTED-PUB-NO: WO 200147533A
BASIC-ABSTRACT:

NOVELTY - Inhibiting (M1) activity of nuclear factor-kappa-B (NF-kB) in a cell, involves contacting the cell with an inhibitor of glycogen synthase kinase-3 (GSK-3).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a non-human transgenic animal model (I) for GSK-3 gene function which has a defect in GSK-3 function;

(2) screening (M2) for biologically active agents that modulate GSK-3b function, involving combining a candidate agent with a non-human transgenic animal comprising a knockout of an GSK-3b gene or an exogenous and stably transmitted mammalian GSK-3b gene sequence, and determining the effect of the agent on GSK-3b function; and

(3) screening (M3) biologically active agents for the specificity of action on GSK-3b function, involving combining a candidate agent with a non-human transgenic animal comprising a knockout of an GSK-3b gene or an exogenous and stably transmitted mammalian GSK-3b gene sequence, and determining the effect of the agent on GSK-3b function as compared to an animal comprising normal GSK-3b function.

ACTIVITY - Antiasthmatic; neuroprotective; antipsoriatic; antiarthritic; antiinflammatory; cytostatic; dermatological; immunosuppressive; antithyroid; antiulcer; vasotropic.

MECHANISM OF ACTION - Inhibitor of NF-kB activity (claimed). No supporting data is given.

USE - M1 is useful for modulating the activity of NF-kB activity (claimed). M1 is useful for treating asthma, systemic lupus erythematosus (SLE), scleroderma, various forms of vasculitis, inflammatory autoimmune myositis, autoimmune thyroiditis, multiple sclerosis, inflammatory arthritis, inflammatory bowel diseases including Crohn's disease and ulcerative colitis, psoriasis, systemic shock and hyperproliferative disorders such as arthritis, inflammation, cancer etc. A non-human transgenic animal model (I) which has a defect in GSK-3 function is useful for assessing the effect of a compound on GSK-3 inhibitor cells, and for the determination of pathways relating to GSK-3.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

3. Document ID: JP 08188600 A

L43: Entry 3 of 4 File: DWPI Jul 23, 1996

DERWENT-ACC-NO: 1996-388604
DERWENT-WEEK: 199639

TITLE: New antibodies inhibiting rat's II-type phospholipase A2 and its fragments - isolating antibody from rat's platelet and releasing phospholipase A2 bound to cells, used in treating e.g. asthma, psoriasis, etc..

PRIORITY-DATA: 1994JP-0340005 (December 29, 1994)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 08188600 A	July 23, 1996		015	C07K016/40

INT-CL (IPC): C07K 16/40; C12N 5/10; C12N 15/02; C12P 21/08; C12P 21/08; C12R 1/91

ABSTRACTED-PUB-NO: JP08188600A
BASIC-ABSTRACT:

An antibody having the following properties and its fragment are new: (1) inhibiting II-type phospholipase A2 (PLA2) isolated from rat's platelet, and (2) releasing the PLA2 bound to cells. Also claimed is a murine monoclonal antibody (MAb) 3A1 or 2R7 produced by a hybridoma 3A1 (FERM P-14654) or 2F7 (FERM P-14653), respectively, or its reduced alkylated deriv. or its fragment. Also claimed is a hybridoma 3A1 or 2F7.

MAb which does not inhibit PLA2 of human, rhesus monkey, dog, rabbit, mouse and cat. Reduced alkylated antibody or its fragment which is prepd. by reducing he S-S linkage between H- and H-chains and/or H- and L-chain and then alkylating with iodoacetonitrile.

USE/ADVANTAGE - The antibody or its fragment (F(ab')2, Fab, or Fab' derived from whole molecule by digestion with trypsin, papain or pepsin) may be used in development of drugs for diseases (e.g. myocardial infarction, cerebral infarction, acute nephropathy, asthma, chronic rheumarthritis, osteoarthritis, septic shock, pancreatitis, psoriasis, multiple organ failure (MOF), acute respiratory distress syndrome (ARDS), Crohn disease and chronic ulcerative colitis, uveitis, respiratory disturbance syndrome (RDS) in new born, bronchopulmonary dysplasia (BFD)) partially or totally mediated by PLA2, using pathogenic rat models. The antibody strongly reacts with rat's PLA2 to completely inhibit its activity at a concn. of 3 micro g/ml (polyclonal). IC50 = 0.1 micro g/ml.

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

4. Document ID: US 5214066 A

L43: Entry 4 of 4 File: DWPI May 25, 1993

DERWENT-ACC-NO: 1993-181825
DERWENT-WEEK: 199322

TITLE: Producing animal model for inflammatory bowel disease - by topical admin. to colonic mucosa of a sulphydryl blocker

INVENTOR: SZABO, S

PRIORITY-DATA: 1990US-0510229 (April 18, 1990)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 5214066 A	May 25, 1993		007	A01N043/36

INT-CL (IPC): A01N 43/36; A61K 31/40

ABSTRACTED-PUB-NO: US 5214066A
BASIC-ABSTRACT:

Prodn. of mammalian idiopathic inflammatory bowel disease in a non-primate laboratory animal comprises topically administering to the colonic mucosa of the animal a compsn. comprising a sulphydryl blocker in an amount effective to produce colonic lesions.

USE - There is provided an animal model for inflammatory bowel disease, including idiopathic ulcerative colitis and Crohn's disease. The model is useful for studying the pathogenesis of chronic ulcerative disease and evaluating protective drugs.